THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

OCTOBER, 1909.

ORIGINAL ARTICLES.

EXOPHTHALMOS AND OTHER EYE SIGNS IN CHRONIC NEPHRITIS.

By LEWELLYS F. BARKER, M.D.,
PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY,
AND
FREDERICK M. HANES, M.D.,
MEDICAL HOUSE OFFICER AT THE JOHNS HOPKINS HOSPITAL, BALTIMORE.

(From the Medical Clinic of the Johns Hopkins University and Hospital.)

IT is the purpose of this communication to direct attention to the frequent occurrence of exophthalmos in chronic nephritis, and to advance the view that the exophthalmos of chronic nephritis is very analogous to that of exophthalmic goitre, being but one of a number of evidences of a chronic systemic intoxication.

Exophthalmos can be easily produced experimentally by stimulating the cervical chain of autonomic fibers. According to the work of Landström[1] and MacCallum and Cornell,[2] this exophthalmos is due to the contraction of a cuff of plain muscle which, attached to the septum orbitale anteriorly and to the equator of the eyeball posteriorly, acts as an opponent of the recti muscles of the eye and produces exophthalmos when it is tonically contracted. This cuff of plain muscle is under the control of the autonomic fibers passing to the eye through the cervical autonomic chain.

[1] Ueber Morbus Basedowii, Thesis, Stockholm, 1907. The Pathogenesis of the Eye Signs of Exophthalmic Goitre, Editorial Jour. Amer. Med. Assoc., 1909, lii, 476, 6.

[2] The Mechanism of Exophthalmos, Med. News, Oct. 15, 1904.

It seems unquestionably true that toxins produced by certain diseases are capable of stimulating the autonomic system and producing, among other effects, exophthalmos. This toxic effect upon the autonomic nerve fibers is most familiar to us in exophthalmic goitre. In Basedow's disease the bodily functions under the dominion of the autonomic system are profoundly affected, owing apparently to overstimulation. Thus, the tremor, tachycardia, exophthalmos, sweating, vasomotor dilatation, and the evidences of irritation of the central nervous system are most probably accounted for by the assumption of chronic systemic intoxication, the exact nature of which need not concern us here.

Undoubtedly every physician of experience has occasionally observed exophthalmos and other eye signs in cases of chronic nephritis. It is surprising, therefore, that not one of the leading systematic treatises upon nephritis which we have been able to consult, makes mention of exophthalmos as a sign of chronic nephritis. It is a not infrequent finding, as we shall presently show. Though we have been observing exophthalmos in chronic nephritis and commenting upon it at the ward visits for several years, the material upon which the conclusions of this paper are based represent the medical service of the Johns Hopkins Hospital with chronic nephritis since January 1, 1909. Our total number of cases is too small to permit of very accurate statistical deductions, but they are valuable in that the patients have been studied with especial reference to the occurrence of exophthalmos and other ocular conditions in chronic nephritis.

Among the total admissions of thirty-three cases of chronic nephritis during the first four months of 1909, 16 (48.4 per cent.) showed exophthalmos. The exophthalmos varied greatly in degree, as did the gravity of the nephritic process in the various individuals; indeed, it may be stated that those cases presenting evidences of serious intoxication (suburemic or uremic symptoms) most frequently showed exophthalmos and one or more of the allied ocular signs—anisocoria, von Graefe's, Moebius', or Stellwag's sign. Exophthalmos has been an obvious sign in all of the cases of chronic nephritis which have died in the Johns Hopkins Hospital since January 1, 1909, seven in number. We have also observed that the cases of chronic nephritis showing albuminuric retinitis during this period have invariably shown exophthalmos, with one or more of the other ocular signs to be spoken of presently. It should be emphasized that exophthalmos is but one of several ocular signs which are frequently present in chronic nephritis.

We have already referred to the work of Landström in demonstrating the presence of a cuff of plain muscle attaching the eyeball anteriorly to the septum orbitale. This author thinks that a spastic condition of this plain muscle—the opponens recti—satisfactorily accounts for the widening of the lid slits (Stellwag's sign),

dissociation of the movements of the ball and lids (von Graefe's sign), and the break of convergence of the eyes (Moebius' sign). All of these classic signs of exophthalmic goitre may be present in a maximum degree in chronic nephritis without the thyroid showing any apparent involvement. The conclusion lies very near, that in both diseases a chronic systemic intoxication affecting the autonomic system is the causative factor in the production of the ocular manifestations. In our series of 16 cases von Graefe's sign was positive in 11 (68.7 per cent.), Stellwag's was positive in 13 (81.3 per cent.), and Moebius' sign was positive in 7 (43.7 per cent.). The pupils were noted as unequal in 5 cases (31 per cent.), and albuminuric retinitis was observed in 8 cases (50 per cent.).

Exophthalmos was associated with arterial hypertension (pressure above 160 mm.) in 12 of our 16 cases, and in 2 cases which may be instanced (Ray C. and Robert W. B.) a maximum degree of exophthalmos was associated with the maximum arterial tension shown by the patient; as the blood pressure fell the eyes became less prominent.

We are not of the opinion, however, that the exophthalmos is due to chronic hypertension. They may exist quite independently, and certainly only a small percentage of cases having arterial hypertension show exophthalmos. R. Sattler[3] has described exophthalmos in some cases of hemorrhagic retinitis with hypertension due to cardiac, renal, and hepatic lesions. It is far more reasonable to assume that the arterial hypertension and the eye signs mentioned are but evidences of poisoning by perhaps separate toxins. It is well known that uremia may develop in a patient whose blood pressure is not increased, and it seems very probable that in chronic renal insufficiency several toxins are present in the blood manifesting themselves in various ways.

Illustrative Cases. Case I.—Annie P., aged thirty-five years, black, laundress. Admitted November 30, 1908; discharged improved April 12, 1909.

Complaint. Shortness of breath and swelling of feet.

Family History. Negative.

Past History. Patient had rheumatism in winter of 1906–07 for three months, which seems to have been of the acute articular type. Patient gets up two or three times each night to urinate.

Present Illness. Dates from April, 1908, when she was admitted to the Johns Hopkins Hospital with dyspnœa, cough, frothy expectoration, and swelling of feet. A diagnosis of aortic and mitral insufficiency was made. After her discharge, in 1908, she remained fairly well for a time, but recently all of her many symptoms have returned in exaggerated form.

Physical Examination. It was noted that the eyes were prominent (Fig. 1), pupils were equal, moderately contracted, reacted slug-

[3] Arch. Ophthal., New York, 1885, xiv, 190 to 195.

gishly, and lids were puffy. There was a well-marked von Graefe's sign. There were signs of aortic and mitral insufficiency in the heart and effusion in the right pleural cavity. The patient had marked anasarca.

She gradually improved, and was discharged when able to walk around without much dyspnœa.

FIG. 1.—Case I.

The urine varied in specific gravity from 1010 to 1020, and in color from pale yellow to orange. The quantity was reduced, as a rule. Albumin was constantly present, ranging from 0.5 to 10 grams per liter by Esbach. Rather numerous hyaline and finely and coarsely granular casts.

The blood pressure averaged about 120 mm. Hg., reaching 140 occasionally.

CASE II.—Ray C., aged twenty-two years, single, Russian Jewess, a shop girl. Admitted January 1, 1909, complaining of twitchings of the right arm and hand and difficulty in speaking.

The patient had been admitted to this hospital also in 1904, when a diagnosis of hemichorea was registered. At that time the urine on four examinations was as follows: Pale yellow in color, specific gravity from 1008 to 1012, trace of albumin, and upon one occasion a few finely granular and hyaline casts. No exophthalmos was noted during this admission. The patient was discharged greatly improved.

The patient's family and past histories are negative except for frequent attacks of tonsillitis which were quite severe, necessitating the removal of the tonsils one year ago.

The attack which brought the patient to the hospital this time began November 1, 1908, with inability to write due to involuntary jerkings of the right arm and hand, and this muscular twitching has

since involved the face and tongue so that speech has been difficult. The patient also complained of headache and pain over the heart.

The physical findings upon admission were as follows: The eyes were markedly protuberant and the lids and conjunctivæ slightly œdematous (Fig. 2). Well-marked Moebius, von Graefe, and Stellwag signs. The heart was slightly enlarged, extending 11.5 cm. to the left in the fourth intercostal space. There was a loud, rough, well-transmitted systolic murmur, with an accentuated pulmonic second sound. The blood pressure upon admission was 245 mm. Hg.

Dr. Bordley examined the patient's eyes and noted a marked œdema of the disk and retina, obscuring most of the vessels on the border of the disk, with several small hemorrhages and white plaques scattered over both fundi. The changes were thought to be unquestionably of nephritic orgin.

FIG. 2.—Case II.

Three weeks after admission the patient developed a severe multiform erythema, which persisted for ten days. On February 10, she complained of excruciating headache and nausea, with vomiting, and at eleven o'clock had a general convulsion. This was followed by three similar convulsions during the afternoon. The patient responded well to treatment, and gradually returned to her former condition. Since these first convulsions the patient has had two similar attacks, with four and ten convulsions, respectively, and unconsciousness lasting from six to twelve hours.

The blood upon admission showed red blood cells, 5,400,000; white blood cells, 13,300; hemoglobin, 90 per cent. The leukocytes have ranged since then from 7000 to 25,000 and the red blood cells have fallen to 4,100,000, hemoglobin to 70 per cent. The urinary picture has been as follows: Total amount from 1 to 1¾ liters; pale

yellow, clear; specific gravity ranging about 1012; no sugar; heavy cloud of albumin, ranging from 2 to 10 grams per liter by Esbach; numerous hyaline and finely and coarsely granular casts.

The blood pressure upon admission was 245 mm. Hg., and since then has been elevated, with an average of about 215, frequently reaching 230.

Exophthalmos (Fig. 2) has been a very constant and striking feature, remarked by all observers. It has varied in degree from time to time, growing definitely less as the patient's condition improved and becoming more marked with each exacerbation.

Case III.—Robert W. B., married, aged fifty-one years, white, a painter. Admitted January 15, 1909. Died January 17, 1909.

The patient had been admitted to the hospital in 1901, and a diagnosis of lead poisoning with peripheral neuritis was then registered. He complained on present admission of severe pain all over the body, and headache.

Family History. Negative.

Past History. Patient says he has had malaria several times. He is subject to severe headaches and has been as long as he can remember. Eye sight has been failing steadily for the last five years. For the last two years he had been suffering with attacks of palpitation and orthopnœa. Gastric and intestinal disorder with vomiting and diarrhœa have been rather frequent of late. He has had to get up several times each night for the past five years to urinate, and passes his urine frequently during the day. The patient has been a heavy smoker, a constant beer drinker, and he very often drinks whisky before breakfast. In 1901 he suffered from a severe peripheral neuritis due to lead poisoning, and was treated in the Johns Hopkins Hospital. At that time the specific gravity of the urine was 1010, with a trace of albumin and a few hyaline casts.

Present Illness. For the past six months the patient has been unable to work much, and has felt very feeble and weak. During this time he has had frequent attacks of abdominal pain, has been constantly constipated, short of breath on exertion, and subject to orthopnœa. One week before admission he was confined to his bed with very severe headache, constipation, and was at times irrational. He passed large amounts of urine during the day and rose from one to seven times at night to urinate.

Physical Examination. Patient in a rather lethargic, torpid condition, respirations 16 to a minute, very deep, and with groaning expirations. Muscular twitchings of the arms noted on admission. The patient was thin, poorly nourished, very anemic looking, had pale mucous membranes and a dry, harsh skin. The eyes were very prominent and staring, the scleræ were visible above and below the iris. The right eye was more prominent than the left. The pupils were equal and reacted normally. There was marked dissociation of the lids, and the eyeball convergence was very poor. The heart

was slightly enlarged, with marked arteriosclerosis of the peripheral vessels. There was a double wrist-drop present.

Upon the day following admission the patient had a general convulsion. It was followed by two convulsions a few hours later. Upon ophthalmoscopic examination there was a well-marked albuminuric retinitis, with œdema, exudate, and hemorrhages in both eyes. The retinal arteries were tortuous and small.

The blood showed 3,000,000 red cells, 11,600 white cells, 35 per cent. hemoglobin. Grawitz granules were demonstrated in the red blood cells. The blood pressure upon admission was 160 mm. Hg., and this fell to 110 at time of death. *It was noted that the exophthalmos,* so marked upon admission, was much less marked at the time of death.

The urine upon two examinations was noted as light yellow; specific gravity, 1012; albumin positive, with large numbers of granular casts, pus cells, and epithelial cells.

The diagnosis of chronic nephritis with acute exacerbation following saturnism and chronic alcoholism was made.

Autopsy, No. 3155. Anatomical diagnosis: Extreme grade of acute and chronic diffuse nephritis; œdema of the brain; concentric hypertrophy of the heart; bilateral bronchopneumonia; pleural œdema; acute pericarditis; fatty degeneration of the liver; anemia.

Case IV.—Jos. B., married, aged forty-eight years, white, a tailor. Admitted January 13, 1909. Discharged February 25, 1909, improved.

Complaint. Cough, pain in back.

Family History. Negative.

Past History. Negative except that for some years he has had to get up two or three times at night to urinate. Drinks five or six glasses of beer daily, two glasses of whisky, and gin occasionally.

Present Illness. Began four weeks ago with a cold and cough and pain in back. His head has ached violently and his eye sight has been growing worse. He has had anorexia and polyuria for the past four weeks.

Physical Examination. Patient was very pale and drowsy. Slight dyspnœa, 28 to minute. The eyes were noted as very prominent and staring, lids somewhat œdematous and inflamed. Right pupil was larger than the left, both reacted to light and accommodation. The heart was slightly enlarged; sounds clear. During the examination the patient became violently delirious and had to be placed in a restraining jacket.

Dr. Randolph, upon ophthalmoscopic examination, noted a high grade of arteriosclerosis of the retinal vessels with several hemorrhages in both fundi; also marked exophthalmos, the left eye being more prominent than the right, and a high grade of peripheral arteriosclerosis.

There were 5,200,000 red blood cells, 11,200 white blood cells,

80 per cent. hemoglobin. The blood pressure upon admission was 240; fell to 140 under continuous use of sodium nitrite.

The urine upon admission was pale yellow, clear, increased in quantity; specific gravity, 1012; marked trace of albumin with moderate number of finely granular and hyaline casts.

As the blood pressure fell the urinary output became greatly decreased, being as low as 150 c.c. in twenty-four hours. The specific gravity averaged 1020, going as high as 1032. The hyaline and granular casts persisted.

The patient left the hospital greatly improved, and a diagnosis of chronic nephritis with uremia was registered.

CASE V.—Oscar T. H., married, aged forty-six years, white. A musician. Admitted March 2, 1909. Died March 5, 1909.

Complaint. Shortness of breath.

Family History. Unimportant.

Past History. The only item of importance is that the patient says he has to get up every hour during the night to urinate. Voids a large quantity during the night, two quarts he thinks. He does not urinate very much during the day. Of late he has had to get up only three or four times during the night. Patient has drunk beer and whiskey to excess, and has been a heavy smoker.

Present Illness. Began two years ago, when patient became short of breath while blowing a cornet as he was marching up hill. This shortness of breath has grown markedly worse, and he had to give up blowing musical instruments. About a month ago he began to be giddy and dizzy upon attempting to work, and this symptom has persisted. His feet have been swollen for the past two weeks.

Physical Examination. Revealed a pallid, dyspnœic man with typical Cheyne-Stokes respiration. Dr. Thayer noted that the radials were thickened, feeling like small cords. The temporals were markedly arteriosclerotic. The eyes were distinctly exophthalmic, and in both fundi there were exudate and hemorrhages together with neuroretinal œdema, presenting a picture of albuminuric retinitis. The heart was enlarged 15.5 cm. to the left and 5 cm. to the right. There was a gallop rhythm to the heart, but no distinct murmurs were audible. The blood pressure ranged from 230 to 200 mm. Hg. There were numerous fine rales at the bases of both lungs. The liver was easily palpable, and there was shifting dulness in both flanks. Dr. Thayer summed up the case as follows: Marked Cheyne-Stokes respiration; œdema of lungs; enlarged heart from high blood pressure; large liver; œdema of back and legs; albuminuric retinitis, and the case was diagnosticated as chronic interstitial nephritis.

There were 5,800,000 red blood cells, 8600 white blood cells, 87 per cent. hemoglobin. The urine was not increased in amount. It was pale yellow and clear; specific gravity, 1010; heavy cloud of albumin; numerous coarsely granular and hyaline casts.

Patient sank into coma and died three days after admission.

Autopsy, No. 3179. March 5, 1909. Anatomical diagnosis: Extreme arteriosclerosis with almost complete obliteration of many of the principal arteries, including the renals, mesenteric, hepatic, coronaries, cœliac, etc.; extreme hypertrophy of the heart; fibrous myocarditis; atrophied kidneys; degenerative changes of the kidneys; chronic passive congestion of the lungs.

CASE VI.—J. J. McF., male, single, aged thirty-two years, white. A bridge worker, using lead paints a great deal. Admitted February 24, 1909. Died March 3, 1909.

Complaint. Pain in the left side and shortness of breath.

Family History. Negative.

Past History. Unimportant, except that the patient has been a worker in lead paints all his life, has drunk freely of beer and whiskey, and has smoked moderately.

FIG. 3.—Case VI.

Present Illness. Patient says he was well until four days before admission. Went to bed at 10 P.M., and as soon as he got into bed became suddenly short of breath. He was unable to breathe lying down. This attack lasted for half an hour. Pain then developed in the left chest, with a cough. Pain in the left side and cough have continued for the past four days and the patient's bowels have not moved during this time.

Physical Examination. There was marked staring exophthalmos of both eyes (Fig. 3). Von Graefe's and Moebius' signs were well marked. The pupils were equal and reacted normally. The heart presented evidence of a pericardial effusion; a systolic murmur at the apex was heard in addition to a pericardial friction rub. There was a pleural friction rub with slight dulness at each base behind.

There was a tophus in the right ear, which contained sodium biurate crystals, and the patient gave a history of arthritis of the big toes, so that he certainly had gout. Dr. Randolph examined the fundi oculi and noted the presence of albuminuric retinitis in both.

On March 1 the pericardium was tapped and about 30 c.c. of hemorrhagic fluid removed.

Blood Examination. 2,900,000 red blood cells, 18,000 white blood cells, and 48 per cent. hemoglobin. The blood pressure was 170 mm. Hg., and fell to 150 at time of death.

The urine was constantly increased in amount, pale yellow; specific gravity, 1010 upon four examniations; albumin 1.5 to 2 grams per liter by Esbach, and there were hyaline, granular, and waxy casts.

Autopsy, No. 3176. March 3, 1909. Anatomical diagnosis: Chronic diffuse nephritis, small granular kidney; lead poisoning; cardiac dilatation and hypertrophy; arteriosclerosis; organizing hemorrhagic pericarditis with effusion; serofibrinous pleuritis, bilateral with effusion; hemorrhagic bronchopneumonia; œdema and atelectasis of the lungs; gouty arthritis; tophi; encapsulated typical tuberculous foci.

CASE VII.—William B., aged forty-six years, white. A metal polisher. Admitted March 4, 1909. Discharged April 5, 1909, improved.

Complaint. Shortness of breath and pain in back and left side.

Family History. Negative.

Past History. Without bearing upon the present illness.

Present Illness. Began four weeks before his admission with shortness of breath and palpitation. He has been troubled pretty constantly of late with attacks of dizziness. He is dyspnœic upon slight exertion. He is up six or eight times during each night to void. Urine is pale, clear, and greatly increased in quantity. He is troubled with cramps in the calves of his legs at night. He occasionally has numbness of the right arm and side, which he has noticed during the past year. The legs are sometimes swollen in the morning.

Physical Examination. Revealed a well-nourished man, slightly dyspnœic. *The eyes were markedly exophthalmic* (Fig. 4). The conjunctivæ were slightly œdematous. The pupils were small, tending to myosis, reacting to light. There was a positive Moebius' sign. No von Graefe's or Joffroy's signs. The lid slits were widened. Dr. Bordley, upon ophthalmoscopic examination, noted that there was marked retinal arteriosclerosis and typical albuminuric retinitis, as evidenced by neuroretinal œdema, white plaques, and numerous hemorrhages in both fundi. The heart was enlarged, extending 14.5 cm. to left in fifth interspace and 4 cm. to right. The heart was irregular in protodiastolic gallop rhythm, and a fine systolic murmur at apex. There was marked general arteriosclerosis.

Dr. Barker summarized the case as follows: "History of alcohol-

ism, acne rosacea, dizziness, palpitation, dyspnœa, polyuria, and pollakiuria, albuminuria, cylindruria, exophthalmos, gallop rhythm, marked arteriosclerosis. The patient probably has a contracted arteriosclerotic kidney with enlarged heart due to the renal and vascular changes. Quite possibly he has a beginning cerebral arteriosclerosis. The heart is beginning to show mitral insufficiency—probably relative."

Blood. Red blood cells, 4,400,000; white blood cells; 8600; hemoglobin, 70 to 80 per cent.

FIG. 4.—Case VII.

The urine was increased in amount, pale yellow; specific gravity averaged 1012; albumin 0.5 to 2 grams per liter; moderate number of hyaline and finely granular casts.

There was chronic arterial hypertension, the pressure seldom falling below 200 and rising as high as 230 on several occasions. The patient's family (wife and daughter) state that the eyes have grown prominent during the past eight months. The diagnosis of arteriosclerosis, chronic nephritis, and myocardial disease was registered.

CASE VIII.—Jeremiah F., married, aged sixty-four years, black. A cook. Admitted April 1, 1909. Discharged April 28, 1909, improved.

Family History. Negative.

Past History. Without bearing upon present illness. For about a year patient has had frequency of micturition, up three or four times at night to void. The same number of times during the day.

Present Illness. Began March 25, 1909, with an attack of shortness of breath. He had pain in his lungs of an acute cramp-like character, and had a severe cough and orthopnœa. His legs began to swell a week before admission.

Physical Examination. The patient was poorly nourished. The skin was dry and harsh. The eyes showed well-marked exophthalmos and positive Stellwag's, von Graefe's, and Moebius' signs. The right pupil was larger than the left. The pupils reacted very sluggishly to light and accommodation. The heart was slightly enlarged, extending 12 cm. to left in fifth intercostal space, and 4.5 cm. to right. The action was irregular with extra systoles. No murmur. There was marked general arteriosclerosis.

Red blood cells, 3,530,000; white blood cells, 4900; hemoglobin, 60 per cent. The blood pressure was normal.

The urine was pale yellow in color, from 1012 to 1020 in specific gravity, with a trace of albumin and a few hyaline casts. The total daily amount of urine was rather small.

CASE IX.—George H. E., widower, aged fifty-three years. A painter. Admitted March 8, 1909. Died forty-eight hours later.

Complained of pain in stomach.

The *family and past history* contain nothing of importance.

The *present illness* began about January 1, 1909. Patient began to lose his appetite and had dull burning pain in the abdomen. This pain bore no reference to food taken. No vomiting. For the past five months the patient has had to get up several times each night to urinate.

Physical Examination. Patient presented a haggard, pallid, emaciated appearance, with a marked dyspnœa. The eyes were strikingly prominent, giving a staring expression to the face. Stellwag's and von Graefe's signs were present. The left pupil was slightly larger than the right; both reacted to light and accommodation. Both parotid glands were swollen and tender. The mucous membranes were pale, and the skin was harsh and dry. The heart was enlarged, extending 12.5 cm. to left. No murmurs. There was diffuse arteriosclerosis in peripheral vessels. The epigastrium was filled with a tumor mass, which extended 8 cm. below costal margin in right mammary line, 3 cm. above umbilicus in mid line.

The red blood cells were 3,260,000; white blood cells, 7600; hemoglobin, 60 per cent. The blood pressure was 110 upon single examination.

Two urinary examinations were made. The urine was greatly reduced in amount, catheterization being necessary to obtain a specimen. The urine was yellow, specific gravity was 1020, distinct trace of albumin; and contained hyaline finely and coarsely granular casts.

The patient died rather suddenly on the second day in the hospital. No autopsy obtained. The tumor mass in the epigastrium was thought to have been carcinoma.

CASE X.—Herman G. M., aged fifty-two years, white. A bricklayer. Admitted April 5, 1909. Discharged improved April 19, 1909.

Complaint. Shortness of breath.

Family History. Negative.

Past History. Patient had malaria thirty years ago and pleurisy twenty-two years ago. Eight years ago, in April, 1901, patient had a paralytic stroke of the right side, involving face, arm, and leg. He recovered almost completely from this attack. The patient drinks gin and whiskey to excess and chews a plug of tobacco a day. His occupation exposes him to inclement weather and subjects him to hard work.

Present Illness. Began two weeks ago, March 23, 1909, with drowsiness, weakness, and shortness of breath. A week later patient had pain in epigastrium just above umbilicus. Appetite became poor and bowels constipated. For the past three days he has vomited two or three times a day. Patient has had to get up eight or nine times at night to void, and about nine or ten times during the day. His ankles and legs are sometimes swollen.

Physical Examination. Revealed a short plethoric individual, dyspnœic, breathing 36 to minute. The eyes were markedly prominent, producing a distinct stare. There was a fairly well-marked von Graefe's sign. The right pupil was slightly larger than the left, both reacted normally. The heart was enlarged 16 cm. to left in fifth intercostal space, and 4.5 cm. to right. No murmurs. There was a well-marked protodiastolic gallop rhythm. There was very slight œdema of the feet.

No ophthalmoscopic examination was made.

Red blood cells, 5,168,000; white blood cells, 14,200; hemoglobin, 95 per cent. The blood pressure ranged from 200 to 130, remaining most of the time between 180 and 200 mm. Hg.

The urine was reduced in amount to 500 c.c. or less in twenty-four hours, which was probably due to the free purgation and sweating. The urine was reddish yellow in color, averaged 1022 in specific gravity; albumin, 0.25 grams per liter by Esbach on five examinations. There was a moderate number of finely granular and hyaline casts.

Case XI.—Henry R., aged sixty-one years, black. Admitted April 28, 1909.

Complaint. Pain in back.

Family History. Unimportant.

Past History. Patient had diphtheria at fourteen, malaria six years ago, and had cough with shortness of breath for past two years. Chronically constipated. The patient is up three times each night to urinate. Has some difficulty in starting stream. Patient has been a heavy drinker for past ten years, taking eight glasses of beer and two or three glasses of gin or whiskey a day. Usually drinks a glass of beer every morning before breakfast. Has worked hard all his life, and is exposed to the weather a great deal. Thinks he has lost 18 pounds in the past year.

Present Illness. In February, 1909, the patient was taken with sudden sharp pain in the small of his back and became very short of breath. The pain in his back was so severe that he came to the Dispensary and was put in a cast two weeks ago, but without relief. This pain has been present for last two months and is worse on coughing and when he stoops over. Has been constantly dyspnœic and orthopnœic. About a week ago his stomach and legs began to swell.

Physical Examination. Patient is irrational, though not violently delirious. Breathing, 32 to minute; breath, urinous. The eyes are moderately exophthalmotic; lid slits wide; pupils very small and react sluggishly. Von Graefe's sign is positive and convergence is very poor. The mucous membranes are pale, and skin is harsh and dry. The heart is enlarged, extending 15.5 cm. to left in fifth intercostal space, 3.5 cm. to right in fourth intercostal space. There is a loud transmitted systolic murmur at apex and a diastolic murmur in aortic area. The liver is somewhat enlarged. Movable dulness in the flanks and definite œdema of the ankles.

Red blood cells, 2,880,000; white blood cells, 6,800; hemoglobin, 50 per cent. Blood pressure is 140 mm. Hg.

Urine is decreased in amount, orange yellow, specific gravity 1014, and contains hyaline and finely and coarsely granular casts.

CASE XII.—George L. P., married, aged thirty-three years. Admitted to the hospital April 25, 1909, and died the same day. Of importance in the family history is the fact that the patient's father and his sister died of tuberculosis, and both grandparents died with Bright's disease.

Past History. Had scarlet fever at eleven without apparent sequels. History otherwise negative.

Present Illness. Began five months ago. Patient noticed he was passing much more urine than normal, up four or five times each night to urinate. Two months ago patient's eyes began to fail him, objects were blurred, and at times he was almost completely blind. Severe headaches have been present for the last two months. The patient grew gradually weaker, and he suffered a great deal with nausea and vomiting. His condition grew gradually worse until nine days before admission, when he took to his bed and vomited constantly. Since this time his urine has been very scanty.

Physical Examination. Positive findings were as follows: Patient was thin and anemic in appearance. The eyes were very prominent, giving a markedly staring appearance to the face. Stellwag's and von Graefe's signs were well marked. Pupils were equal and reacted to light and accommodation. The mucous membranes and skin were very dry. The heart was enlarged, extending 11.5 cm. to left in fifth intercostal space and 3.5 cm. to right in fourth intercostal space. There was a short systolic murmur at apex. The aortic

second sound was louder than the pulmonary second. There was moderate arteriosclerosis.

Upon ophthalmoscopic examination a marked albuminuric retinitis was revealed, with neuroretinal œdema and many white patches of exudate. Several large hemorrhages in both fundi.

The blood picture was as follows: 4,192,000 red cells, 21,800 white cells, 60 per cent. hemoglobin. The blood pressure was 120 mm. Hg.

The urine was decreased in amount; specific gravity, 1012; pale yellow in color; albumin in large quantity, Esbach, 4 grams per liter, and there were fairly numerous granular casts.

CASE XIII.—Daniel H., aged fifty-five years, married. A farmer. Admitted April 29, 1909, complaining of weakness. Died in coma May 1, 1909.

The *family* and *past history* are without bearing upon the present illness. The patient gave a history of rising once each night to urinate.

The *present illness* began a year ago. The patient noticed that he was gradually growing weaker. Exertion caused him shortness of breath, which symptom has grown constantly more marked during the past year. About one month ago he noticed that his sight was failing, and since then has been unable to read ordinary print. There has been no œdema of the feet and ankles. He has been getting up two or three times at night to urinate, but thinks the total quantity voided has not been greatly increased. Ten days before admission the patient sank into a coma from which he could be easily aroused. Four days ago the patient had a general convulsion, since when he has been unconscious, and was brought into the hospital in a comatose condition.

The positive physical findings were as follows: Patient comatose, and at times there are rapid twitchings of the right arm. There is a coarse tremor of the hand. Breath is urinous. Mucous membranes are pale. The right pupil is larger than the left. Both react normally. The eyeballs are markedly prominent and there is a well-marked von Graefe's sign. The right eyeball seems more prominent than the left. Upon ophthalmoscopic examination there was a most marked neuroretinal œdema, arteries tortuous, and scattered throughout both fundi numerous hemorrhages and white plaques, these being especially numerous around the macular region. An albuminuric retinitis was diagnosticated.

The blood showed 3,920,000 red blood cells, 10,680 white blood cells, 73 per cent. hemoglobin. The blood pressure upon admission was 210 mm. Hg., and sank gradually to 130 at time of death, four days after admission.

The urine was noted as light yellow, clear; specific gravity, 1010; heavy cloud of albumin with a few granular casts.

The patient did not respond to treatment, and died four days after admission.

CASE XIV.—Annie J., aged fifty years, widow.

Diagnosis. Chronic nephritis; arteriosclerosis; had four former admissions, with diagnosis of chronic nephritis in each. The eyes were staringly prominent, marked Stellwag's and von Graefe's signs.

Heart 13.5 cm. to the left in the fifth intercostal space. No murmurs.

Urine, 1005 to 1020; pale yellow; contains albumin and a few granular casts. Blood pressure from 240 to 180 mm. Hg.

Autopsy, February, 1909. Anatomical Diagnosis: Arteriosclerosis; obesity; chronic diffuse nephritis; chronic fibrous myocarditis; cardiac hypertrophy and dilatation; chronic passive congestion of viscera.

CASE XV.—Henry P., married, aged 55 years.

Diagnosis. Arteriosclerosis; myocarditis; chronic nephritis; mitral insufficiency. Eyes noted as exophthalmic. No mention of other ocular signs.

Heart 14 cm. to the left in fifth intercostal space. The first sound at the apex is followed by a rough transmitted murmur. The patient is very dyspnœic.

Urine, 1010 to 1020; albumin, 1 gram per liter by Esbach; a few hyaline and granular casts; pale yellow is the color. Blood pressure, 160 to 190 mm. Hg. No ophthalmoscopic examination. Discharged improved.

CASE XVI.—Henry P., aged fifty-four years.

Diagnosis. Chronic nephritis; cirrhosis of the liver. Death and autopsy.

The patient gave history very typical of nephritis. Up three or four times each night to urinate; anemic.

The eyes were noted as quite prominent; ocular movements were good.

Urine, light yellow; specific gravity, 1010 to 1012; contains albumin and many granular casts. Blood pressure, 160 mm. Hg.

Autopsy. Anatomical diagnosis: General arteriosclerosis; chronic diffuse nephritis; pulmonary œdema; cirrhosis of liver.

THE NATURE OF THE ARTERIOSCLEROTIC PROCESS.[1]

By J. George Adami, M.D., F.R.S.,
PROFESSOR OF PATHOLOGY IN MC GILL UNIVERSITY, MONTREAL.

Surely all of us who are active pathologists have been impressed by the fact that the more autopsies we perform the larger does arteriosclerosis loom as the fundamental morbid process in the majority of deaths after the age of forty years. If trauma, malignant disease, and infections, such as pneumonia and the rarer tuberculosis, be eliminated, we are left with that vast mass of cases of cardiac incompetence, aneurysm, chronic bronchitis and emphysema, cerebral apoplexy, and chronic Bright's disease; and the more we study these cases the more it is borne in upon us that in these the terminal event is but an outcome of the one common underlying process of arterial disease. At the same time, the more carefully we study the arteries in these cases the more we recognize that we deal with an almost protean series of disturbances: now it is the upper portion of the aorta, the ascending limb, or the arch that shows the most pronounced and obvious lesions; now the abdominal aorta; in other cases the aorta is relatively free from sclerotic change, but its larger branches show irregularities, this with or without pronounced involvement of the smaller arteries, which in their turn are not equally affected; at times the renal, at times the cerebral, at times the arteries of the extremities are the seats of most advanced change. In the second place we find that the lesions are not all of one order, so much so that it may well be debated, as it is debated, whether one common name should be applied to the series. I am free to acknowledge that this name arteriosclerosis is not wholly satisfactory. Marchand[2] has recently recommended that of atherosclerosis, but this, like the older atheroma, is inadequate, in that it implies that the development of ἀθήρη, or a porridgy degeneration, is inevitable. Something might be said for this suggestion had we to deal only with the aorta and the larger vessels, although even in these there are developed sclerotic states—I refer to those of syphilitic origin—which do not by any means necessarily pass on to an atheromatous stage. (These Marchand would place in a separate class.) It is, however, inadmissible for in the condition as found in the smaller sized arteries the sclerotic change is predominant, and the atheromatous or degenerative is characteristically absent. Again, it may rightly be objected that the atheroma is a secondary and not a primary condition; it is always the secondary outcome of fibrosis. "Arteriocapillary fibrosis," save for its flatulency, has more in its favor

[1] The annual address delivered at a meeting of the Pathological Society of Philadelphia, April 24, 1909.

[2] Verhandl. Deut. path. Gesellsch., 1906.

than atherosclerosis, but again is incorrect, for the capillary changes are not obvious, and, as I shall point out, when present must be regarded as secondary. We come back, therefore, to this, that the term "arteriosclerosis" is the most satisfactory name that has so far been afforded for this series of arterial changes, since sclerosis, which is equivalent to fibrosis, is the condition of commonest occurrence in the series of included conditions.

What, then, are the different forms that we have to take into account? So much has been written upon the subject of late years that I need only refer to the salient points, for doubtless the majority here present have as full a knowledge of this matter as have I who speak. And first as to the main orders of disturbance seen in the aorta and its larger branches—in the vessels, as we may term them, of the elastic type:

1. Most common is the ordinary nodose arteriosclerosis. This shows itself in the slightest cases as a thickening and sclerosis, more particularly at or around the origins of the side arteries, as yellowish, white thickenings notably affecting the origins of the series of intercostal arteries. Later these have grown in size, and some may have coalesced, forming in the opened aorta projecting flattened nodes, and now these may be scattered somewhat irregularly through the aorta, tending to be more abundant and more advanced in the abdominal region. It is this form that, more particularly, passes into the condition of atheroma, and of calcification of the intimal plaques.

2. Almost as frequent is a contrasted order of disturbances, in which clinically we have the most obvious proof of the existence of arteriosclerosis, in the presence of hardened and at times pipe-stem radials; but when we come to examine the aorta we may in the most typical cases find a complete absence of nodose thickening of the intima. In its place there is a diffuse dilatation of the aorta, affecting more particularly the thoracic portion. Instead of being thickened, the aortic wall appears to be thinned and the increased volume of the tube tends to show itself not only in breadth, but also in length, the vessel becoming somewhat tortuous; the arch takes a wider sweep, the abdominal section is curved. This I am inclined to regard as the uncomplicated senile type of the disorder. It is further characterized by the presence, in the common iliacs and the carotids, of slight depressions, tending to have their long axis situated transversely, in fact, by the very reverse of the sclerotic nodosities, a giving way taking the place of thickening of the wall. And the question may well be asked: Should this be included? It is not a sclerosis, but merely a degeneration. This I freely admit, but would urge that the condition of the whole arterial tree has to be taken into consideration in making our diagnosis, and if we do this we find that in these cases the smaller arteries are markedly sclerotic, with localized areas of intimal fibrosis, with hypertrophy and fibrosis

of the middle coat and often with well pronounced peri-arterial fibrosis. The sclerosis of the radials and other middle-sized arteries, often so prominent in these cases, is paradoxically not a sclerosis proper; it is not necessarily due to fibrosis of the intima or other coats. Sometimes there is a diffuse thickening of the intima of moderate grade, but that is not the cause of the hardening. As Dr. Klotz[3] has most serviceably pointed out, the essential change in vessels of this type is a calcification of the media, which I may add, following Russell,[4] is apt to be preceded by a marked hypertrophy of the middle-coat. For convenience, I would speak of this order of arteriosclerosis as Moenckeberg's sclerosis, Moenckeberg[5] having been the first to make a full study of what is the most striking feature, namely, the areas of giving way and slight sacculation seen in the main branches of the aorta due to degeneration of the media. But, doing this, I would impress upon you that uncomplicated examples of this order of disturbance are relatively uncommon. Quite the commonest condition in elderly people is a combination of these two types, having occasional nodes or plaques of sclerosis or atheroma in the aorta, more particularly in its abdominal portion, though by no means necessarily confined to this region, with this faint saccular giving way in the common iliacs and carotids, suggesting strongly that the same order of events leads now to the one, now to the other development; that, in fact, they are diverse manifestations of a common state.

3. The third great type is the syphilitic. The aortic manifestations of this form are in the broad features so much of the same order as those of ordinary nodose arteriosclerosis that it is only during the last generation and, indeed, during the last seven years that the distinguishing features have attained general recognition. Thanks to the labors of Döhle[6] of Kiel and other pupils of Heller, and of Chiari,[7] we now have no difficulty in making the distinction, if not always by the naked eye, then surely under the microscope. The seat of election in the first place is in the ascending aorta and arch, and here the nodes are liable to lie in groups. These in their earlier stage are large and, if I may so express it, succulent, with a semi-translucent or hyaline appearance; characteristically they have little tendency to atheromatous and calcareous change, but, on the contrary, exhibit a later scarring or central depression with some puckering. The reason for this is found upon microscopic

[3] Calcification of the Aorta in Rabbits after Injection of Adrenalin, Jour. Exp. Med., 1906, viii, 325; Calcification of the Media in Arteries of the Elastic Tissue Type, Jour. Exp. Med., 1906, viii, 330; The Relationship of Experimental Arterial Disease in Animals to Arteriosclerosis in Man, Jour. Exp. Med., 1906, viii, 304; Experimental Arteriosclerosis, Brit. Med. Jour., December, 1906; Experimental Work-Arteriosclerosis, Montreal Med. Jour., March, 1908, and Centralbl. f. allg. Pathol., 1908, xix, 535.

[4] Arterial Hypertonus, Sclerosis, and Blood Pressure, Edinburgh, 1907.

[5] Virchow's Archiv, 1903, clxxi, 141.

[6] Inaug. Diss., Kiel, 1885.

[7] Präger med. Woch., 1806, xxxi, No. 12; Verhandl. Deut. path. Gesellsch., 1904.

examination. The primary disturbance here is subacute mesaortitis, with small-celled infiltration around branches of the vasa vasorum and absorption of the elements proper of the media. Coincidently there is overgrowth of the intimal tissues, and when, as a result, the deeper portions of the overgrowth exhibit necrotic change and degeneration, then the underlying inflammatory granulation tissue advances new capillaries into the necrotic area. The result is an absorption of the degenerated material, replacement by cicatricial tissue, shrinkage, and scarring. So far as my observations go, there is a striking lack of vascularization and replacement in the ordinary nodose arteriosclerosis. It does not occur during the necrotic atheromatous stage; I have encountered it when there is already pronounced intimal calcification and the necrotic change is extending into the inner layers of the media.

Here in connection with syphilis, and yet more markedly, there is to be observed that same paradox seen in connection with the Moenckeberg type; while the syphilitic virus leads to the more pronounced intimal overgrowth, at the same time it leads characteristically to the opposite condition of lack of intimal overgrowth, thinning of all the coats, and aneurysm production. It will be familiar to you that modern workers, with the exception of von Hansemann, ascribe from 60 to 85 per cent. of aneurysms to syphilis. How are we to reconcile these opposing results?

These are the main types. Acute changes do not come under the heading of arteriosclerosis, nor do simple degenerative changes without proliferation. The fatty streaks of the intima seen often after acute infection, are not here included, although these and certain other intimal changes may throw light on certain phases of the process. To them I shall refer later.

Turning now to the other arteries, we may, as regards arteriosclerotic change, divide them into the muscular arteries and the arterioles. As regards the former, the same changes, the ordinary thickening of the intima, the giving way of the Moenckeberg type, and the syphilitic changes may be seen as in the aorta, with, however, one or two modifications which would seem to be a function of relative size. Thus we do not encounter anything like the same extent of atheromatous and degenerative change. Compared with the size of the artery, the areas of intimal thickening may be extreme; nevertheless, in absolute size they do not compare with the plaques seen in the aorta. And it may be reasonably suggested that degeneration when it occurs is due to cutting off of the nutritive lymph supply by the development superficially of layer after layer of dense new connective tissue, and that in general the size of those plaques in the arteries (save in the larger cerebral vessels and the coronaries) is not sufficient to inhibit the percolation of lymph and arrest of nutrition of the more central areas. This does not, however, mean that there may not be extreme calcification, more especially in the

middle sized arteries; only that calcification affects, more particularly not the intima, but the media.

This brings us to the second point of difference, namely, that in these arteries the media exhibits much more obvious alteration. Where there are relatively large plaques of intimal thickening there beneath them, even with the naked eye, the media is seen to be notably thinned. But, on the other hand, it may exhibit diffuse thickening from hypertrophy of its muscle tissue, with little obvious intimal change. On the other hand, there may be degeneration of the muscle and replacement by connective tissue; in some regions this may be associated with giving way and dilatation of the arterial lumen with associated intimal thinning; in others there is seen a more or less diffuse thickening and overgrowth of the intima, suggesting compensatory change. Passing to the arterioles, we find, it is true, the same series of changes, but here the relationships are significantly altered. There has been active debate as to their significance and relative frequency. In one order of cases the muscular hypertrophy of the middle coat has been the dominating change. This is especially well seen in certain kidneys, so much so that sundry observers have laid stress upon this as the cardinal change. In another series fibrosis is the most evident alteration—fibrosis of the *intima*, fibrosis replacing largely the *muscular* tissue of the *media*, fibrosis and pronounced overdevelopment of the *adventitia*. So that other observers have waved the muscular change to one side and regarded the fibrotic change as the all-important condition.

Yet another state has also been dwelt upon, particularly in association with the infectious type, as seen in tuberculosis, syphilis, and chronic glanders, namely, the active cellular proliferation of the intimal endothelium leading to an obliterative endarteritis. It is still unsettled whether this proliferation is directly connected—as Baumgarten believes it to be—with the production of the intimal fibrosis—whether, that is, the endothelium directly gives origin to the connective tissue overgrowth of these arterioles, or whether the processes are distinct. Provisionally I incline to Baumgarten's opinion.

How now are we to harmonize and bring into a common scheme these various lesions? What, in short, is the fundamental underlying change—if there be any—common to be different conditions? The answer to this question can be approached in two ways—either by an exhaustive analysis of the factors seen to be in action in the various types of arteriosclerosis; or by taking one form in which the causative factors have been most fully determined and applying the results gained from the study of this form to the other cases, to observe whether any common general principles are capable of application to all. Both, I believe, lead surely to the same end result, and let me confess that I have been puzzled as to which method to employ. It seems to me, however, that the former would con-

sume too much time; it would demand a preliminary discussion of the minute anatomy and of the physiology of the arterial tree, of the regulation of the blood pressure, and the influence of contraction of the arterioles upon the aorta and the aortic conditions. It is true that, employing the other method, reference must be made to most if not all of these matters, but I imagine that it will be less wearisome if I take up a concrete case and then proceed to apply the results gained from that to a consideration of the other cases.

Now undoubtedly we possess the clearest and most indubitable data regarding the syphilitic form. There is no question nowadays that the primary lesion in syphilitic aortitis is a granulomatous condition or small-cell infiltration immediately surrounding the terminal vasa vasorum in the media; the primary condition is, as Chiari describes it, a syphilitic mesaortitis, and this involves in the first place the outer half or two-thirds of the aortic media. Certain of the vasa only are affected. Here, just as in acute mycotic aneurysm, as shown by Dr. J. McCrae's[8] study of a case published from our laboratory, the infective agent gains entrance into and lodgment in particular vasa. And here it may be noted that several observers have discovered the spirochete in the aortic lesions. With this infiltration there is, as Dr. Klotz points out in an article shortly to be published, a remarkable dissolution of the elements of the media. They seem literally to melt away. This explains the thinning and depressions which may occur, so that, as Aschoff points out, placed against the light these areas are semitranslucent. To what an extent this process may extend is well shown in the accompanying specimen (Fig. 1) which Dr. Klotz has been so good as to permit me to demonstrate to you. This has been stained to demonstrate the elastic tissue. You will observe that in the region where there is the most extensive small-cell infiltration the elastica has completely disappeared.

I will not say that this dissolution is specific. I do not believe that it is. I am inclined to think that it may be the outcome of local inflammation, due to more than one cause, with swelling, degeneration, and necrosis of the cells of the involved area. But undoubtedly the most frequent cause is syphilis. Here, therefore, is the onset of the syphilitic or mycotic type. It will be readily understood how such extreme destruction of the media, the main sustaining coat of the aorta, leads on to aneurysm. In fact, we may say that it is universally acknowledged nowadays that degeneration of the middle coat is the dominant cause of aortic aneurysm. But such extreme destruction of the middle coat as here shown is the exception and not the rule; so again in syphilitic aortitis aneurysm production is the exception; what is far more common and widespread is the pro-

[8] Jour. Path. and Bact., 1905, x, 373.

duction of intimal thickening. How is this to be explained? I doubt if there be any more obvious example of compensation in the whole of pathology; degeneration of the one coat, the media, is accompanied or followed by overgrowth of the other, the intima; nay, more, as Dr. Klotz's example illustrates very beautifully, and as I confess I had not previously realized, in connection with the aorta there may[9] be a coincident and very pronounced hypertrophy of the adventitia (Fig. 1).

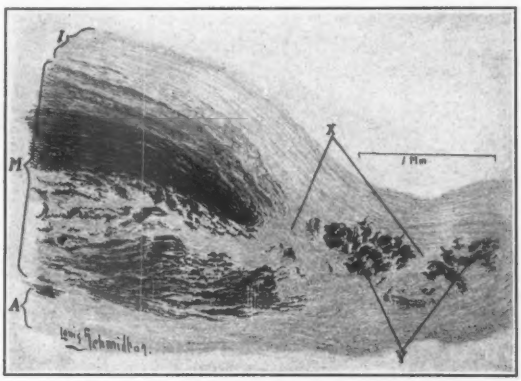


FIG. 1.—Section from a case of syphilitic mesaortitis stained by the elastic-tissue stain to demonstrate the extraordinarily degenerated and diseased appearance of the elastic tissue through syphilitic mesaortitis. It will be seen that at *X* the elastic fibres have completely disappeared; at *Y* they are reduced to irregular masses. There is some definite hypertrophy, both of the intima, *I*, and of the adventitia, *A*, but this has been insufficient to prevent a certain giving way of the wall. (From a specimen prepared by Dr. Klotz).

Certain points stand out very prominently regarding this intimal thickening. In the first place, it exhibits none of the ordinary earmarks of an inflammation. There is no formation of granulation tissue properly so-called; no new vessels; no small-cell infiltration, and, notwithstanding this, the new connective tissue formation attains to large proportions. The intima, it is true, is in itself non-vascular; it receives its nourishment by diffusion from the plasma within the aorta, as, indeed, would seem to be the case with the innermost layers of the media. But if we take any other non-vascular region, the cornea for example, and induce in that an inflammation by any of the accepted methods—by the injection of bacteria or the employment of caustic agents—within a short time new vessels make their way to the injured area from the periphery. Save as a late event, as already noted, we do not encounter this in the intimal thickening in syphilis

[9] Since this address was delivered, J. H. Wright and O. Richardson have also called attention to this thickening of the adventitia with extensive necrosis of the media. Boston Med. and Surg. Jour., 1909, clx, 539.

even when there is an active granulation tissue formation in the media. This is a very remarkable fact; the intimal overgrowth is essentially and purely hypertrophic. The internal elastic lamina constitutes as it were a barrier. We do not deal with an endarteritis, and Virchow was wrong in labelling it with this name; as again have been Thoma and other later writers (Fig. 2).

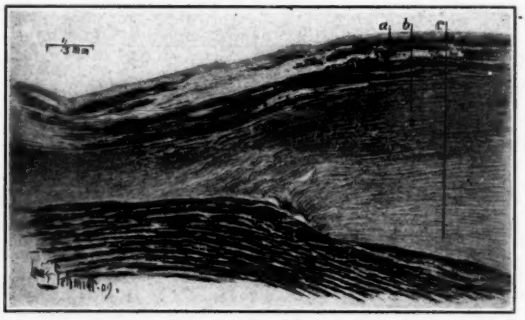


FIG. 2.—Section of an aorta from syphilitic mesaortitis with some narrowing of the media and great overgrowth of the intima. It will be observed that the layers of new tissue of the intima show a regular arrangement with absence of vessels and of small-cell infiltration. The outer layers, at *a*, in this case exhibited pronounced fatty change. The intermediate layer, *b*, took on a slight stain with the elastic tissue reagent, and under the high power this was seen to be due to the fine fibrils of what, from their arrangement, were young connective tissues, taking on the elastic tissue stain. Deeper down, at *c*, there was a complete absence of this reaction. The media, on the contrary, while narrowed, stained deeply with this stain. (From a specimen prepared by Dr. Klotz.)

But if an hypertrophy, how is it to be explained? We owe to Thoma,[10] in connection with ordinary arteriosclerosis, the first clear recognition that this is a compensatory hypertrophy—that in ordinary arteriosclerosis there is first a giving way of the media, and that the intima proliferates as a consequence. Thoma sought to explain this overgrowth as of nutritive origin, as due to alteration in the rate of blood flow at the region of bulging of the arterial wall. For myself, I must confess that I have never been able to grasp his explanation; what is more, I have never met anyone who has pretended to do so. Thirteen years ago, in an address before the New York Pathological Society, I afforded an explanation.[11] That also was not comprehended at the time. For example, Professor Mott[12] wholly mistook its drift. But I am more and more convinced that that explanation was rational, and, what is more, recent studies of a different nature have afforded a brilliant demonstration of its

[10] Virchow's Archiv, civ, and subsequent volumes.

[11] On the Relationship between Inflammation and Sundry Forms of Fibrosis, Middleton Goldsmith Lecture, Med. Rec., 1896, pp. 469 and 505; see also Discussion upon Arteriosclerosis, Brit. Med. Jour., December, 1906, ii.

[12] Allbutt's System of Medicine.

correctness. I laid down then that this regular development of layer after layer of new connective tissue was non-inflammatory, but was of the nature of *strain hypertrophy*—that just as increased pull or strain of the muscles upon their bony insertions leads in athletes to an increased development of the bony ridges, to a localized hypertrophy of bone, so within certain limits, increased tension or pull upon fibrous connective tissue favors its overgrowth, provided that adequate nourishment be afforded at the same time. It is but another example of the law, wholly misunderstood by Weigert, that increased functional activity within certain limits favors growth. I am glad to see that Professor Aschoff[13] explains the progressive increase in the elastic tissue of the aorta as being due to a like work hypertrophy.

I held, and so I still hold, that a moderate local giving way of the media under the pressure to which it is subjected by the blood within an artery causes an increased strain upon the overlying intima, which must undergo a certain amount of stretching, as it now becomes pressed outward to expand over the concavity afforded by the bulging of the media (Fig. 3). Herein is the stimulus to growth, and the growth continues, layer after layer, until not, as Thoma held, the concavity is accurately filled and the lumen restored to the status quo ante, but until the volume of the new tissue and the resistance that this affords to the mean distending force balances the loss sustained by the weakened media. In other words, when the resistance offered by the whole arterial wall at such a locality is equivalent to that of the rest of the aortic or arterial wall, then the strain is reduced and the process of hypertrophy becomes arrested. In these cases the youngest tissue is situated immediately beneath the endo-

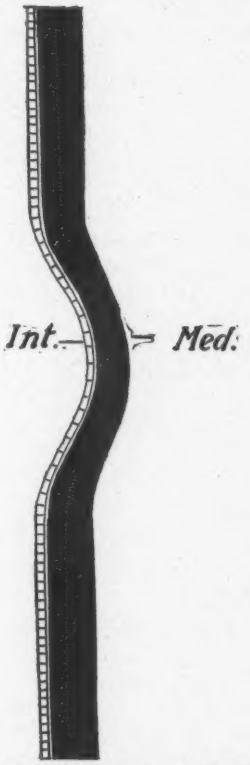


FIG. 3.—Schematic representation of the increased strain brought to bear upon the cells of the intima, *Int.*, when the media, *Med.*, undergoes a localized expansion through relative weakness.

[13] Beihefte zur med. Klinik, 1908, iv.

thelium, the oldest most remote and nearest to the deeper musculo-elastic layer. Unlike what occurs in the ordinary form of arterio-sclerosis, in syphilis this latter layer, so far as I have seen, shows comparatively little hypertrophy.

The convincing demonstration of the accuracy of this view has been afforded by one of Carrel's remarkable transplantation experiments. Carrel,[14] it will be remembered, removed a length of the cats carotid and replaced it by a length of vein from the same animal and of approximately the same caliber; under the increased blood pressure the vein exhibited dilatation, but left in situ for many weeks it presented at the end of the period a remarkable connective tissue hypertrophy of all its coats, so that its eventual thickness was distinctly greater than that of the artery at either end of the transplanted piece. The adequate and, in fact, as I freely admit, improved nutrition to which the vein is subjected, might by some be held to explain the hypertrophy of the intima, but at the same time the more that becomes sclerosed, the less becomes the nutrition of the deeper layers; but all the coats showed the connective tissue overgrowth. Add to this that cases in which increased nutrition, pure and simple, leads to hypertrophy are almost if not entirely wanting. I can bring to mind no single incontrovertible example. There is, I hold, no other satisfactory conclusion than that in this case it is the increased strain thrown upon the venous wall (coupled with adequate nutrition) that leads to the profound connective tissue hypertrophy.

When, on the other hand, the destruction of the media is more widespread and, we must presume, more acute, then, as in aneurysm formation, we encounter not intimal overgrowth, but the reverse. In other words, the expansion of the intima which has wholly lost its main support, the media, is now so great that we pass from the state of *strain* to that of *overstrain*, and now with the overstrain in place of hypertrophy we tend to get atrophy.

We thus arrive at the following conclusions regarding the effects of syphilis upon the aorta: (1) The primary disturbance is a granulomatous, inflammatory degeneration of the media. (2) This leads to a local giving way of the aorta. (3) If this be moderate it results in a strain hypertrophy of the intima and of the adventitia, with the development of a nodose intimal sclerosis. (4) If it be extreme, there results on the contrary an overstrain atrophy of the intima and aneurysm formation. (5) The intimal nodosities are here not of inflammatory type and are non-vascular, although, with the progresive laying down of layer upon layer of connective tissue on the more internal aspect of the intima, the earlier and deeper placed layers of new tissue gain less and less nourishment, and so are liable to exhibit fatty degeneration and necrosis. (6) These products of necrosis exert a chemiotactic influence upon the nearby

[14] Jour. Exp. Med., 1908, x, 130.

vessels of the medial granulation tissue, with, as a result, (*a*) a secondary and late entrance of new vessels into the early and deeply placed atheromatous area; (*b*) absorption of the necrotic products; (*c*) replacement by granulation tissue; (*d*) contraction of the granulation tissue; and (*e*) depression and scarring of the sclerotic nodules so characteristic of syphilitic sclerosis.

Can we apply these deductions to ordinary non-syphilitic nodose arteriosclerosis and to the Moenckeberg's type already noted? I think we can, or otherwise, and this is the crucial point, we can demonstrate that the ordinary arteriosclerosis of advancing life exhibits a primary giving way of the media, and we can do this both histologically and by experiment. First, as regards the histology, a most interesting study has been published recently by Professor Aschoff regarding the progressive changes that are undergone by the aortic media in the course of life. I will not go into all the details, but merely epitomize his main conclusions. In infancy the elastic laminæ of the media stand out sharply defined, well separated from each other by the muscle layers, which are well developed. There is, that is to say, sharp alternation of muscle and elastic layers. From childhood there is to be observed a slowly progressive increase in the elastic elements of the media. Not only do the individual lamellæ seen in cross-sections become thicker, but also they afford an increasing number of fine secondary filaments feathering off from these and crossing the muscle layer, so that now they are no longer sharply defined, but more ragged upon cross-section. This progressive increase attains its maximum at or about the age of thirty-five, and from now on for the next fifteen years the condition is relatively stationary. After fifty, according to Aschoff, there is to be observed a slowly progressive atrophy of the elastica. The media becomes obviously thinner and presumably weaker.

Independently, Dr. Klotz has been studying the same subject for some years and for the last twelve months Mr. Foster, under his direction, has examined a long series of aortas, from those of infancy up to those of advanced age. His work is on the eve of publication. Again, not to enter into details, I may say that these observers are wholly in agreement with Professor Aschoff's findings up to the age of thirty-five and in practical agreement up to that of fifty. But, as Klotz has already pointed out on more than one occasion, the subsequent degenerative process is one showing itself histologically not so much in connection with the elastic lamellæ as with the intervening muscle. This from the age of thirty-five onward begins to exhibit fatty degeneration, although it is only after fifty that, as a rule, this becomes at all marked. And now one or both of two processes may occur. Either, as shown by Klotz's admirable studies, the fatty gives place to a calcareous infiltration, fine calcareous granules appearing in the course of the muscle fibers—a sign of necrosis of these fibers—or they undergo complete absorption. Per-

haps most frequently both processes are to be made out. As a result the intervening spaces between the elastic lamellæ become smaller and smaller, and often there is afforded the appearance of thick lamellæ due to the approximation of two or more, which can be resolved under higher magnification. With this, whereas in the young aorta the lamellæ are wavy, due to the contraction of the intervening muscles fibers, now they are relatively straight. It is this muscular atrophy rather than atrophy of the elastic tissue that thus would seem to be the main cause of the thinning of the aortic media. I do not in the least mean to say that the elastica is unaffected in this process. At times the calcareous degeneration affects it also. Everything indicates that coincidently it *loses its elasticity*, becomes more rigid and more expanded as the result of internal pressure; it is unapt to return to its former length.

It is further to be noted that the calcification is unequally distributed. Frequently in Dr. Klotz's specimens of the Moenckeberg type it is most marked, and the thinning of the media is coincidently most pronounced, in the walls of the shallow sacculations already noted. The presence of the sacculations show that here are the regions of greatest weakening. But it is frequent to find that when there is marked nodose thickening of the intima then also the underlying aortic media is thinned. This, it is true, is most evident not in the aorta, but in its branches—in the carotids, the splenic artery, the coronaries, etc. In all of these there may be an extraordinary overdevelopment of the fibroid intima and an equally striking expansion and giving way of the media. All must be familiar with specimens showing this condition. On the other hand, it has to be admitted freely that there may be well-marked intimal nodes of the aorta when the underlying media shows no recognizable thinning. Apart from possible exceptions to be subsequently noted there is an explanation to be afforded for this state of affairs—an explanation along the lines of Thoma's well-known observations upon the disappearance of the projecting intimal nodosities when the aorta is filled with melted lard under pressure. This is most rapidly afforded by the accompanying diagram (Fig. 4), which represents a length of aortic wall, in which the midportion has become weaker than the rest. This portion under internal pressure will be liable to give way, and, while thus bulging, will of necessity exhibit a definite grade of thinning, with compensation or strain overgrowth of the intima tending to fill the concavity. If, as occurs postmortem, the internal pressure be removed, then the natural elasticity of this midportion of the wall, which has not been wholly lost, will result in a disappearance of the concavity, the wall now appearing to be of normal thickness of media with a projecting nodosity of the intima. The same will naturally appear in sections prepared under the same conditions.

Let us now turn to the experimental evidence. The active dis-

cussion as to whether the lesions caused by adrenalin, barium chloride, nicotine, and other drugs in the rabbit are to be regarded as in any way equivalent to arteriosclerosis as seen in man, will be familiar to all of you, and many of you may have come to very definite conclusions upon the matter. Certainly these lesions do not resemble the ordinary nodose arteriosclerosis, but, as all must admit, the constant feature is a giving way of the middle coat, and this to such an extent that at times definite aneurysmal pouches are produced, at others and frequently those more shallow sacculations, scarce to be designated as aneurysms proper. As Klotz and K. Ziegler[15] independently pointed out, the appearance is identical with that noted by Moenckeberg in his type of arterial change. Medial degeneration and giving way is the dominant lesion of adrenalin disturbances. I took occasion to point out at the discussion on Arteriosclerosis, at the Toronto meeting of the British Medical Association in 1907, that the reason for the lack of intimal overgrowth might well be the acute development of the medial degeneration, resulting in rapid expansion of the intimal sheath and overstrain rather than strain, and I suggested that less extreme degeneration would be found to result in a more typical arteriosclerosis with intimal hypertrophy. Since then Dr. Klotz, by a different method, has demonstrated the truth of this surmise on my part. He has set up arterial disease in the rabbit without the use of any drug whatsoever, and that by merely suspending a (previously) healthy well-grown buck for three minutes daily by the hind legs—head downward. This naturally caused *pro tem.* an unaccustomed blood pressure in the thoracic, cervical, and cranial vessels, a reduced pressure in the abdominal and other arteries of the hinder end of the body. After this is continued for one hundred to one hundred and twenty days the heart is found greatly hypertrophied, the arch and the thoracic aorta are dilated, with very distinct thinning of their walls; the abdominal aorta, on the other hand, is unaffected; the carotids are

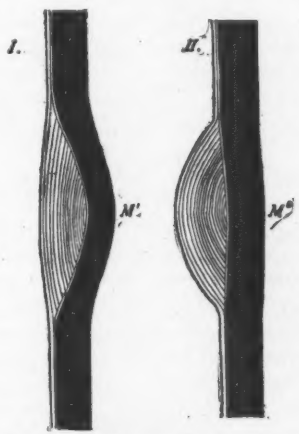


FIG. 4.—*I*, media weakened at M' with overgrowth of intima filling in the depression. *II*, with postmortem rigor and contraction of the muscles of the media and removal of the blood pressure from within, the stretched media at M'' contracts; the intimal thickening thus projects into the arterial lumen.

[15] Ziegler's Beiträge, 1905, xxxviii, 229.

larger than normal, and—this is the important point—they exhibit a typical irregular intimal sclerosis. There is pronounced connective tissue thickening of that intima.

These results can only be explained in one way, namely, as the effects of the daily temporary suddenly increased blood pressure brought to bear upon the vessels of the anterior half of the body.

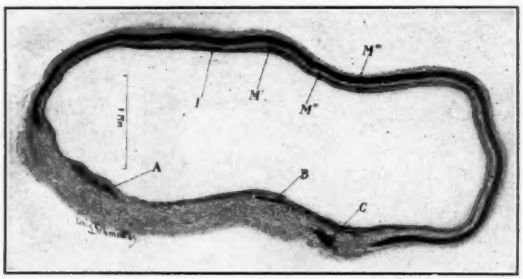


FIG. 5.—Section through the aorta of a rabbit that had been suspended for three minutes daily by the hind legs, for one hundred and thirty days, to demonstrate dilatation and relative absence of intimal proliferation. Around the greater part of the organ there is pronounced thinning of the media with calcareous degeneration of the middle layer (M''). This dilatation and thinning are less marked round about one-third of the organ, and here there is a slight grade of patchy hypertrophy of the intima at *A* and *B*. This hypertrophy here particularly involves the deeper musculo-elastic layer. At *C* there is beginning calcification of the media. (From a specimen stained with Sudan III and silver nitrate by Dr. Klotz.)

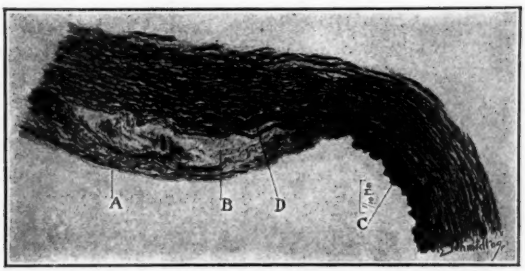


FIG. 6.—Section from the carotid of the same rabbit (higher magnification) exhibiting at *A* well marked nodular sclerosis of the intima. At *B* the deeper layers of the hypertrophied intima are already showing degenerative changes. *C*, normal intima. *D*, media. (From a specimen by Dr. Klotz).

That has resulted in diffuse dilatation of the larger vessel, the aorta, without intimal overgrowth, and in pronounced intimal overgrowth of the smaller vessels, the carotids. Here for the first time, I think, we have an absolute demonstration that one and the same cause is responsible for the two orders of conditions, the demonstration that the arterial thinning of the Moenckeberg type and the intimal thickening of ordinary (senile) arteriosclerosis are diverse mani-

festations of a common process. The greater circumference and the relatively slighter resisting or accommodative force of the aortic wall due, I may say, to its anatomical structure, has led to overstrain and dilatation, and as a consequence absence of intimal proliferation; the lesser circumference of the carotids and their proportionately greater resisting strength has been followed by a relatively slighter dilatation, tending to be localized to particular areas, and here as a result there has been strain, not overstrain, of the intima and a resultant connective tissue hypertrophy. I must add in justice that another Canadian worker, Dr. Harvey, of Toronto, continuing work begun under Professor J. J. MacKenzie, has independently, in the laboratory of Professor Dixon, of Cambridge, England, brought about the same diffuse dilatation of the aorta and cardiac hypertrophy by temporal digital compression of the rabbit's aorta extending over long periods. He does not, however, in his communication call attention to the coincident sclerotic change in the smaller vessels.[16]

Here then we arrive at a common order of events in the two great types of aortic sclerosis—the syphilitic and the ordinary nodose forms. In both we find weakening of the media as the primary disturbance; in both, if this be above a certain grade, there is pronounced giving way without intimal compensation; in both, if below this grade, a strain hypertrophy of the intima is manifested, leading to the production of intimal thickening. Only, in syphilis the giving way of the media is the more extreme, and so we more frequently encounter advanced aneurysmal formations as contrasted with the shallower sacculations of the Moenckeberg type, and in this, also, occurring as a rule earlier in life, there is a more exuberant regenerative power exhibited by the intima.

This view, it will be seen, demands that strain or pressure is the cause of the intimal overgrowth in both orders of disturbance; it demands also that the giving way of the media in the first place is due to a local weakening sufficient to render the arterial wall unable to resist the outward thrust of the column of blood. Nay, more, Klotz's experiment shows that increased intravascular pressure alone may be the cause of the medial degeneration and weakening in the first place, of the giving way of the arterial wall in the second, and of the intimal hypertrophy in the third. We are brought thus face to face with the time honored problem—to what extent is heightened arterial pressure—or, as Sir Clifford Allbutt would seek to have us call it, *hyperpiesis*—a factor in arteriosclerosis?

From the data already afforded, it is evidently not an essential factor in the syphilitic cases. Undoubtedly when present it may

[16] Personal communication from Professor MacKenzie and Sir Clifford Allbutt. I was of the impression that Harvey's results had been published, but can find no reference in the Index Medicus or elsewhere.

accelerate the process in these, but infection is here the primary agent in causing the atrophy and absorption of the media.

As regards senile arteriosclerosis, the observations of Aschoff, Klotz, and Foster show that after fifty there is to be determined a physiological atrophy of the media; obviously the degeneration of the muscular layers and the further indications of change in the elastic lamellæ indicate a weakened media. It is not necessary in these cases to demand an increased blood pressure. The normal blood pressure must eventually be adequate to cause giving way of the media with or without consequent intimal hypertrophy.

Granting this for senile cases, then for the presenile arteriosclerosis, we are, from Klotz's experiment, forced to recognize that high intra-arterial pressure, and this alone, is sufficient to bring about a nodose arteriosclerosis with all its possible dangers. That experiment affords the experimental proof of the danger of heightened blood pressure, which from practical experience clinicians have so learnt to fear—and this in a perfectly clear manner, with no ambiguities or possibilities of lack of appreciation of other factors. High pressure, and high pressure only, can be invoked to explain the results which I have demonstrated to you.

We then reach the following conclusions, namely: That *like results may ensue when, on the one hand, the artery has undergone weakening and the blood pressure is normal, and when, on the other hand, the artery has no preliminary degeneration of its media and the blood pressure is above the normal.* What is of importance is that, as shown by this experiment, it is not necessary to have a blood pressure which is permanently above the normal; an acute rise for a short period frequently repeated will bring about the result.

I would greatly like to enter into a discussion of the mechanism of increased arterial pressure in relationship to peripheral arterial sclerosis, but have already taken so much of your time that I can venture at most to suggest the headings. I would call your attention to the fact that it is the arterioles through the body and their tonus that determine the existence of the normal blood pressure; that heightened blood pressure in the larger vessels means increased contraction of the arterioles of important areas; that thus to changes in the arterioles and smaller arteries we must eventually look for the explanation of hyperpiesis; that it is by contraction of the arterioles that adrenalin, digitalin, barium chloride and the other raisers of blood pressure produce their effects; that in this contraction we have the elements necessary for the establishment of a vicious cycle; that, as shown by Leonard Hill, the arteries react normally to increased internal pressure not by dilatation, but by contraction; that thus the higher the blood pressure the more contracted do the smaller arteries tend to become; and that, if the heart responds to the increased work thrown upon it, the higher still becomes the blood pressure. Along these lines it will be seen that rise of blood pressure tends in the

first place to throw increased work upon the musculature of the smaller arteries, tends, that is, to cause their hypertrophy; if the rise is progressive, then this hypertrophy gives place to exhaustion, muscular degeneration, and dilatation of the arteries (and arterioles). As a result strain hypertrophy of the intima shows itself with thickening, and it may also be of the adventitia, resulting in chronic periarteritis. And now, with continued degenertion of the medial muscle in those muscular arteries, fibrosis of the media may also show itself. I would thus regard muscular hypertrophy of the arteries and fibrosis of the different coats as different stages in one and the same process. Whether these peripheral changes are the more marked, or the central, depends, I would suggest, upon the relative resisting power of the elastic and the muscular arteries of the individual respectively.

Lastly, it may be asked, have I included here all the forms of arteriosclerosis and intimal hypertrophy? I certainly believe that I have referred to the more important, though I have not exhausted the forms. There is, for example, a form which Aschoff would term functional sclerosis, seen in the ovaries and uterus, in the ovaries after menstruation and ovulation, in the uterus after menstruation and placentation. The sclerotic change here may be extreme, and again is of a hypertrophic and regenerative type—so much so that in the uterus there may be encountered, as shown by Sohma,[17] the development of what is practically a new artery within the old, with endothelial, intimal, muscular, and even what corresponds to an adventitial coat. Here, I would hazard, the same priniciple is at work. The extreme congestion and dilatation of these arteries in menstruation and placentation leads to an overstrain of their walls with consequent overexpansion and degenerative weakening, to such an extent that at the conclusions of these physiological events, when there is no longer a call for dilatation of the arteries, they still remain expanded. Nor is there any adequate mechanism on the proximal side (toward the aorta) to cut off fully the blood supply. There is still, therefore, a certain amount of strain exerted upon the arterial walls from within, and this leads now to secondary overgrowth.

Secondly, Dr. Klotz has shown experimentally, and Saltykow[18] has confirmed, that certain bacterial toxins, *e.g.*, the diphtheritic, lead to medial degeneration, others, like typhoid toxins, have no recognizable effect upon this coat, but induce a primary intimal proliferation. I freely accept the existence of such primary intimal overgrowth. I have to confess, however, that I have some doubts as to its frequency as a cause of extensive arteriosclerosis in man. When it is at all well developed, then I would point out, the superficial intimal overgrowth

[17] Archiv f. Gynäk u. Obst., 1908, lxxxiv, 84.
[18] Ziegler's Beiträge, 1908, xlii, 187.

cuts off the due nutrition of the musculo-elastic layer, and, indeed, of the innermost layer of the media; degeneration and weakening of these is the natural result, and there is established a vicious circle; the greater the intimal thickening the greater becomes the weakening of the lower intimal layer, and so of the arterial wall; nor is the full picture of arteriosclerosis attained until both events are present.

The same picture is afforded in the arterioles. My former colleague, Dr. Duval,[19] now professor of pathology in Tulane University, then pathologist to the Montreal General Hospital, published certain most interesting studies upon the effects of attenuated glanders bacilli upon the vascular system of the rabbit. In these he demonstrated most clearly that these bacilli induce an active proliferation of the endothelium of the smaller arteries, and he gives a figure of a later stage in which a well-marked sclerotic node of the intima has associated with it a bulging and thinning of the media. If these two pictures represent successive phases of one process—and of that I am not wholly convinced—it must still be admitted that medial weakening, whether by the direct action of the glanders toxin or by arrest of nourishment of the media through the intimal hypertrophy, is an essential part of the process. Endothelial proliferation alone does not constitute arteriosclerosis.

Lastly, and perhaps of the greatest present importance, there is the consideration of the part played by the deeper or musculo-elastic layer of the aortic intima in the arteriosclerotic process. Following the lead of Jores,[20] the majority of the recent German writers on the subject lay stress upon the degeneration and hypertrophic processes found in this as being the fundamental change in arteriosclerosis. Here I have an objection to make of the same order as my criticism of the use of the term atherosclerosis, namely, that aortic disturbances are dwelt upon to the exclusion of the concomitant changes in the arteries of muscular type. It is only in the larger branches of the aorta—those of the intermediate type—that this layer continues to be a factor of any importance. As we pass into the smaller arteries its development becomes so slight that it cannot possibly be a factor of the first order.

In the aorta its fully developed structure—of longitudinally arranged plain muscle fibers and abundant elastic fibrillæ—renders it akin to the media. At most, the longitudinal arrangement of its elements suggests that its function is to oppose elongation rather than dilatation of the vessels in which it is present. Simple hypertrophy must connote increased strength of the arterial wall; degeneration must signify weakening of the wall as a whole. Now, significantly this layer is curiously apt to exhibit degeneration. Why this should be so is not wholly understood. I would suggest that its position relative to sources of nutrition is largely at fault. The

[19] Jour. Exp. Med., 1907, ix, 241.

[20] Ziegler's Beiträge, 1902, xxxii, 146.

internal elastic lamellæ of the media largely cut it off from nutrition from without; the plasma from the interior of the vessel has to pass through and be acted upon by the more superficial layers of the intima before reaching it. It has no independent source of nutrition, and more particularly if the superficial layers of the intima have undergone thickening, the factors favoring degeneration are clearly greatly increased. The striking picture that this layer presents in the earlier stages of degeneration—the swollen condition of its cell elements, the œdema of its interstitial substances, the diffuse soapy and fatty change that it undergoes—all, I am inclined to lay down, tend to arrest the attention and tend thus to a neglect of the concomitant medial and superficial intimal changes. And this point of view is supported by the existence of degenerative changes in the layer unaccompanied by any other vascular disturbance. In cases of burns and of acute infections, and that even in the young, it is common to meet with fine fatty streaks in the aorta. And these streaks indicate a fatty degeneration of, in the main, this musculo-elastic layer. It is natural to imagine that here we have the first stage in the arteriosclerotic process. I am, however, convinced that the two have no necessary association. I would ask anyone who doubts this to follow the example of Drs. J. McCrae and Klotz in our service at the Royal Victoria Hospital, and make a note upon the postmortem report every time that these fatty streaks are present. It will surprise him to find how common they are; how frequently they are present in the quite young; how little obvious relationship they have to the arteriosclerotic state.

I do not deny that a weakening of the musculo-elastic layer weakens the whole arterial wall, and thus may be a factor favoring subsequent intimal hypertrophy. I only urge that this is not nearly so frequent or so important as is a weakening of the media.

Thus we revert to our main conclusions that the dominant primary event in the arteriosclerotic process—syphilitic, senile, or functional—is a localized or it may be a diffuse weakening of the arterial wall and especially of the media. This induces increased strain upon the remaining coats; and if this be not excessive, that strain leads more especially to connective tissue overgrowth and the development of the characteristic lesions of arteriosclerosis.

In the foregoing remarks I have repeatedly referred to the work accomplished in our Montreal laboratories, and more particularly to the studies of Dr. Klotz. When the chief of a laboratory quotes the work of those under him, it is usual to infer that the work is his under another name—that he has inspired the researches. In justice to my colleague, I may say that in this instance this most decidedly is not the case. I have quoted Dr. Klotz's work so frequently because I have followed it with very deep interest and know intimately its progressive developments, and further because

each step has, to my mind, confirmed and expanded the conclusions I reached long years ago regarding the essential nature of the arteriosclerotic progress. I appreciate too highly the originality of Dr. Klotz to claim more than having been fortunate in suggesting to him that line of inquiry into the nature of calcification which he carried out with so much distinction and of which much of his later work is, as it were, a natural evolution. I should be glad to regard this address, and to have this address regarded, essentially as a vehicle for bringing forward in a connected manner the observations of my colleague and my interpretation of them, which must not necessarily be thought to be his interpretation.

SOME CONDITIONS AFFECTING THE DISCHARGE OF FOOD FROM THE STOMACH.

By Carl A. Hedblom,

and

Walter B. Cannon, M.D.,

George Higginson Professor of Physiology in the Harvard Medical School, Boston.

The purpose of this investigation was to determine as accurately as possible, under otherwise normal conditions, the influence of some of the commoner physical, chemical, and biological factors on the movements of the stomach and on the rate at which it discharges food into the intestine. The method employed was that used by one of us in studying the passage of different foodstuffs from the stomach.[1] The food used, except when otherwise specified, was 25 c.c. of mashed potato, with which was mixed 5 grams of subnitrate of bismuth. The consistency, which depends upon the amount of water added, was always as nearly uniform as could be judged by the eye and by manipulation. It will appear, however, from observations described below, that within the limits involved variations of consistency have no appreciable effect on the rate of gastric discharge of this food. Full-grown normal cats, deprived of food for twenty-four hours previous to the experiment, served for observation. In all the experiments, except those with fluid food, the animals were placed on the holder and fed from a spoon with no special difficulty. At regular intervals after feeding, observations were made by means of a fluorescent screen illuminated by the *x*-rays; and the dark shadows made by the food were traced in outline on transparent paper laid over the screen. The aggregate

[1] Cannon, Amer. Jour. Med. Phys., 1904, xii, 387.

length of these shadows measured the relative amount of food in the intestine at the different times of observation.

The method has been demonstrated to be above serious criticism.[2] During the first two hours, when the aggregate length of the food-masses in the intestine is most significant as an indication of the rate of gastric discharge, the intestinal content is usually not great, and can therefore be traced with only slight liability to error.

In establishing the normal rate of discharge as a basis for comparisons, the possibility of subjective differences, the personal equation, is involved. That Magnus[3] has recently used the method above described, with no essential variations from the original results, indicates that the personal equation need not be great. In order to estimate the extent of a possible deviation in our work, however, the original observations on mashed potato were repeated by one of us (Hedblom). The two tables which follow show a close agreement in the results independently obtained.

Hours after feeding	½	1	2	3	4
Aggregate length, in cm., of food masses in small intestine (Cannon)	9	30	43.5	28	22
	10	39	53.5	28.5	26
	9.5	22	36	36.5	30
	9	30.5	39	8	7
Average (4 cases)	9.5	30.5	43	25	21

Hours after feeding	½	1	2	3	4
Aggregate length, in cm., of food masses in small intestine (Hedblom)	10	32	42	31	31
	7	24	43	34	26
	11	29	43	33	23
	13	27	38	33	30
Average (4 cases)	10	28	41.5	32.5	27.5

After thus determining that a resonably close agreement existed between the results secured by the use of the method by different persons, we undertook a study of the effects on the rate of gastric discharge, of variations in the consistency of the food, the presence of gas in the stomach, extremes of temperature, different degrees of acidity, massage, irritation of the intestines, and fatigue.

THE INFLUENCE OF CONSISTENCY. A number of investigators have studied the effect of varying the water content of the food, as well as the rate of discharge of various liquids. In some of these researches animals with duodenal fistulas were used; in others the stomach tube was relied upon to determine the rate of discharge. Moritz,[4] who used the fistula method, found that water begins to leave the dog's stomach immediately after being swallowed, and that the stomach is almost emptied in fifteen to thirty minutes. In one case milk was discharged with similar rapidity; in another case, when the milk coagulated, the stomach was not emptied for

[2] Cannon, loc. cit., p. 391.

[3] Archiv f. die ges. Phys., 1908, cxxii, 210.

[4] Ztschr. f. Biol., 1901, xlii, 572.

one and a half hours. These results Moritz confirmed on man. He states that water leaves the stomach of man as quickly as it leaves the stomach of the lower animals; but that beer, milk, bouillon, and thin soup remain longer than water. Even with the least fluid of the substances he used—a thick soup—the stomach was practically empty in an hour. These observations, that liquids leave the stomach rapidly, are confirmed by the work of Hirsch,[5] Penzoldt,[6] Schüle,[7] and Roux and Balthazard.[8] These studies, however, are concerned almost entirely with liquids and not with the discharge of a certain food in various consistencies. To obtain information regarding the effects of varying consistency and other mechanical factors on the gastric discharge, observations were made on more or less viscous samples of potato, on hard particles mixed with the food, and on coarse graham bread.

1. *The Influence of Variations in the Viscosity of the Food.*—For this study potato was used. It was baked in order to drive off most of the water. Two series of observations were made. In the first series no water was added; the potato when mixed with bismuth subnitrate and ready for feeding was very thick and doughy. In the second series water was added until the mixture was of the consistency of thin gruel. The volume fed in all cases was 25 c.c. The results with these extremes of consistency should be compared with the results when potato of the standard consistency (intermediate between the extremes) is fed. The following were the figures obtained.

Potato, thick doughy consistency.

Hours after feeding	½	1	2
Aggregate length, in cm., of food masses in small intestine	5	11	28
	16	28	49
	17	28	41
	13	40	54
	5	17	35
Average (5 cases)	11	25	41.5

Potato, thin gruelly consistency.

Hours after feeding	½	1	2
Aggregate length, in cm., of food masses in small intestine	14	20	35
	9	17	31
	15	22	40
	17	20	40
	18	37	42
Average (5 cases)	14.5	23	37.5

As the curves in Fig. 1 show graphically, the rates of discharge of the same kind of food with fairly widely varying consistencies are nearly the same. Indeed, the rates of discharge do not differ among themselves enough to permit any noteworthy significance to be attributed to the differences in consistency.

[5] Centralbl. f. klin. Med., 1893, xiv, 75.
[6] Deut. Archiv f. klin. Med., 1893, li, 567.
[7] Ztschr. f. klin. Med., 1896, xxix, 49.
[8] Arch. de phys., 1898, xxx, 90.

Further evidence that consistency alone is not a prominent factor in determining the rate of gastric discharge is furnished by Moritz,[9] who observed wide differences in this rate when various fluids of practically the same consistency were given. Thus, water passed from the stomach much faster than beer, and considerably faster than bouillon.[10] On the whole, the conclusion seems warranted that while liquids leave the stomach very rapidly, more or less dilution of a food predominantly carbohydrate has but slight effect in modifying the rate of gastric discharge.

Protein food leaves the stomach more slowly than carbohydrate. The explanation for this phenomenon is that the union of protein with the acid of the gastric juice delays the appearance of a sufficiently strong acid reaction of the contents near the pylorus and thereby delays the first opening of the pylorus.[11] If protein food is diluted with water there is evidently, in a given amount of the food, less protein to unite with the acid than would be present if the same amount were given undiluted.

Lean beef of standard consistency gives the following average figures:

Hours after feeding	½	1	2	3	4
Cm. in small intestine	1.5	2.5	16	22	24.5

When lean beef, shredded and mixed with water to a thin gruelly consistency, was fed, the following figures resulted:

Hours after feeding	½	1	2	3	4
Aggregate length, in cm., of food masses in the small intestine	0	11	20	23	29
	7	9	30	30	28
	0	20	32	33	32
	5	8	22	32	30
	6	22	35	38	37
	0	8	29	39	32
	8	21	33	38	36
Average (7 cases)	3.5	14	28.5	33	32

A comparison of the figures in the foregoing tables and the curves in Fig. 2 shows that the dilution of the protein food, and the reduction thereby of the material uniting with the acid of the gastric juice, tends toward a more rapid discharge of the protein from the stomach.

Schüle[12] states in connection with his investigation of the discharge of liquids that fluid foods seem to increase the motility of the stomach. An inference to the same effect can be drawn from

[9] Loc. cit., p. 590.

[10] The slow emergence of beer and bouillon, compared with water, can be explained by the effect of these fluids in stimulating the flow of gastric juice (see Pawlow, The Work of the Digestive Glands, London, 1902, p. 138 et seq.) and the resultant closure of the pylorus through the acid reflex from the duodenum.

[11] Cannon, Amer. Jour. Phys., 1907, xx, 292.

[12] Ztsch. f. klin. Med., 1896, xxix, 73.

Moritz's statement: "Solid food causes a checking of gastric peristalsis, which overcomes the stimulating effect otherwise exerted upon it by water." We have never noted any significant changes in peristalsis due to variations in consistency.

2. *The Influence of Hard Particles in the Food.* There are numerous clinical reports of foreign bodies swallowed accidentally or wilfully by children and demented persons, and in some instances by normal adults. Eckold[13] reviews more than seventy such cases in which objects of the most varied size and character were swallowed. When they remained long in the stomach more or less serious derangement of gastric functions resulted, followed by malnutrition and in a large percentage, by death. The stomach may evidently function, though imperfectly, while containing a foreign body. But these cases represent decidedly abnormal conditions, and do not throw much light on conditions that may be classed as more or less common, such as the presence in the stomach of bone, seeds, and poorly masticated food.

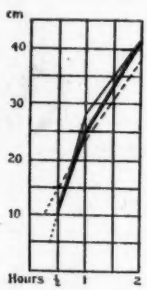


FIG. 1

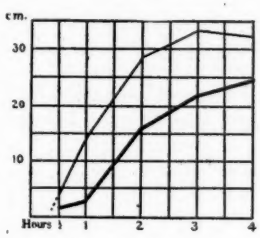


FIG. 2

FIG. 1.—These and the following curves show the average aggregate length of the food masses in the small intestine at designated intervals after feeding. The light continuous line is the curve for potato of standard consistency; the heavy continuous line, for thick doughy consistency; the broken line, for thin gruelly consistency.

FIG. 2.—The heavy line is the curve for lean beef of standard consistency, the light line that for lean beef of thin gruelly consistency.

Few observations as to the relation between hard food masses and gastric discharge have been reported. Schüle[14] states that coarse food remains in the stomach longer than food of smoother consistency. Moritz[15] has described experiments on a dog with a duodenal fistula, in which finely chopped sausage began to leave the stomach in forty-five minutes, whereas coarse unchopped sausage did not begin to leave for two hours, and then appeared at the fistula in a "soaked slippery condition." Cannon[16] reported in his

[13] Inaugural Dissertation, Greifswald, 1896.
[14] Loc. cit., p. 75.
[15] Verhandl. der deut. Naturforscher und Aerzte, 1893, p. 25.
[16] Amer. Jour. of Phys., 1898, i, 359.

first paper on the stomach that hard particles repeatedly pushed up to the pylorus checked the outgo of food from the stomach. The method used in the present investigation permits a more careful testing of this conclusion.

As hard particles, small irregular pieces of starch paste were used. Ten per cent. starch paste, mixed with subnitrate of bismuth, was cut into cubes and set aside to dry. From fifteen to twenty of these dried cubes, 1 to 5 mm. in dimension, were given mixed with the standard potato. In the first four cases reported below the the particles, which had dried for two weeks, were very hard. In the other cases the particles dried only two to four days.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine[17]	4	5	14
	0	3	9
	2	5	7
	0	0	0
	7	13	23
	0	8	22
	0	8	25
	4	13	20
	2	16	21
	1	13	23
Average (10 cases)	2	8.5	16.5

In Fig. 3 the normal discharge is compared graphically with the discharge when the same food, with hard particles added, is fed. As the figures and the curves show, there is a marked retardation of the outgo of food from the stomach when hard particles are present. In the first four cases the outgo was clearly slower than in the others, as if the harder particles had had an inhibitory effect until softened. The checking action of the softer particles, however, shows likewise the working of a mechanical factor in modifying the normal mechanism at the pylorus.

The hard particles with a large bismuth content could be seen on the fluorescent screen as small black shadows against the less dark background of the shadow of the stomach. The to-and-fro oscillations of the small masses in the antrum already described[18] were repeatedly observed. There is a discrepancy between the immobility of these particles in the cardiac end of the stomach, as described in the earlier paper, and Beaumont's report that food circulates down along the greater and up along the lesser curvature. It is conceivable that the difference of observation might be due to a difference in the consistency of the food—that circulation might occur in a fluid gastric content, but not in a semisolid mass.

Starch paste of the consistency of thin gruel, with bismuth sub-

[17] In several cases, notably Case 4, there was an accumulation of gas in the stomach. The presence of the gas tended still farther to check the rate of the discharge.

[18] See Cannon, Amer. Jour. of Phys., 1908, i, 369.

nitrate added, was given. Several (3 or 4) of the hardened starch particles were then swallowed by the animal. In all the experiments the particles lay along the lesser curvature, until gradually carried toward the pylorus. There was no evidence of a circulation of the gastric contents such as Beaumont described.

3. *The Influence of Coarse Graham Bread.* There is evidence that food containing spicules of bran, such as coarse graham bread, may stimulate peristalsis in the large bowel and thus prevent constipation. It was of interest to determine whether gastric peristalsis and the discharge from the stomach are affected by food of this character. There are statements in the literature that no appreciable difference exists between whole meal and white bread in the duration of their stay in the stomach. Penzoldt[19] remarks, however, that light bread remains in the stomach as long as black bread and longer than bread made of coarse meal.

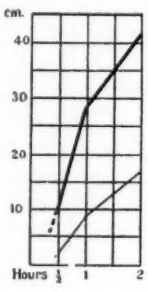


FIG. 3

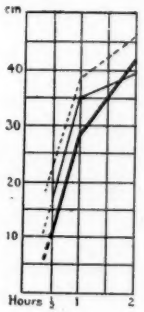


FIG. 4

FIG. 3.—The heavy line is the normal curve for potato; the light line, the curve when hard particles are present in the food (10 cases).

FIG. 4.—The heavy line is the normal curve for potato, the light line that for white bread (7 cases), the broken line that for graham bread (11 cases).

In the observations on the rate of discharge of bread, common white bread obtained at a bakery was used as a control instead of potato. The bread was prepared, however, just as in the observations with potato, and the same proportion of bismuth subnitrate was added. The following are the figures for white bread:

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	10	30	41
	26	40	33
	15	33	30
	20	35	46
	19	42	41
	10	30	36
	16	36	49
Average (7 cases)	16.5	35	39.5

[19] Deut. Archiv. f. klin. Med., 1893, li, 560.

The graham bread fed in the first eight of the following cases was not so coarse as that fed in the last three cases:

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	23	44	48
	30	45	49
	35	42	33
	24	47	48
	16	45	49
	25	24	46
	25	37	44
	19	40	50
	10	33	51
	16	25	44
	25	34	45
Average (11 cases)	22.5	38	46

A graphic comparison of these results with the results when potato is fed is shown in Fig. 4. The curves and the tables reveal a slightly slower rate of discharge for white bread than for potato, and a more rapid discharge for graham bread than for either of the other foods.

In summarizing the effects of the consistency of food on its discharge from the stomach, it may be fairly stated that within limits more or less water added to carbohydrate food does not change the rate of outgo, though dilution hastens the outgo when protein is fed; that hard particles in the food distinctly hinder the discharge from the stomach; and that coarse branny food is forced through the pylorus at a slightly more rapid rate than are similar foods finer in texture.

The Influence of Gas in the Stomach. The common use of carbonated water has led to attention being paid to the effects of carbon dioxide on the functions of the stomach. There is fair unanimity of testimony that this gas stimulates both secretion of hydrochloric acid and gastric peristalsis.[20] That the presence of a body of gas in the stomach might affect the exit of food has apparently not been much considered. Yet with the *x*-rays gastric peristalsis may be seen moving over an accumulation of gas without either churning the gastric contents or propelling them onward. The gas acts as a shield, keeping the walls of the stomach away from the food. We desired to learn what might be the effect of a considerable amount of gas in the stomach on the discharge.

The animals were fed the standard amount of food, and then air was blown into the stomach. In the first experiments the volume of air introduced was measured, but this procedure proved to be of little use, for eructations soon changed the original quantity. In all but the first four of the following cases the air was introduced

[20] See Jaworski, Ztschr. f. Biol., 1883, xix, 443; 1884, xx, 234; Gillespie, Deut. med. Woch., 1887, p. 836; Penzoldt, Deut. Archiv f. klin. Med., 1903, p. 567.

while the animal was under x-ray observation; in these latter cases the distention of the stomach walls, which could be clearly seen, could be easily regulated. In a few instances eructations nearly emptied the stomach of air during the first hour; more air was then introduced until approximately the original volume was restored.

The following results were obtained:

Hours after feeding	½	1	2	3
Aggregate length, in cm., of the food masses in the small intestine	0	6	29	37
	9	34	44	44
	13	27	33	35
	14	24	34	40
	0	25	30	27
	0	30	33	24
	0	17	13	38
	0	17	31	33
	0	0	33	40
	14	26	36	38
	0	10	32	32
	3	6	22	29
	0	11	27	35
	0	10	24	32
Average (14 cases)	4	17.5	30	34.5

These average figures are compared with the average figures for normal conditions in Fig. 5. Examination of the table shows that there is, as was to be expected, a wide variation in the effects produced by the presence of gas in the stomach. In few cases, however, is there any effect except a retardation of the discharge into

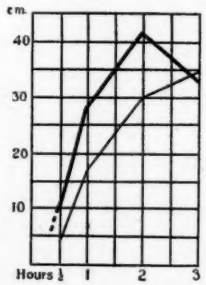


FIG. 5.—The heavy line is the normal curve for potato, the light line that for potato when gas is present in the stomach.

the intestine. This result has been noted repeatedly in other instances in which gas appeared in the stomach spontaneously. Thus, in one case in which fibrin was fed and in which the peristaltic waves could be clearly seen passing over the gas in the stomach the discharge was as follows:

Hours after feeding	½	1	2	3	4	5
Cm. fibrin, when gas present	0	0	0	10	17	22
Cm. fibrin, average of 4 normal cases	4	8	21	29.5	32.5	32

Such cases of spontaneous accumulation of gas seemed to be associated with gastric atony and enfeebled peristalsis. When the air was experimentally introduced, however, peristalsis, when observed, was normal in rate and intensity.

With peristalsis normal, how may the retardation of the discharge from the stomach, noted in the above experiments, be explained? That the distention of the gastric walls prevented them from exerting a direct propelling action on the food mass was distinctly to be seen. In a number of the observations of this series the food occupied relatively the position represented in Figure 6. There was direct contact between the stomach wall and the food only at one surface. Thus the gas prevented the churning of the food by the peristaltic waves and the gradual propulsion of the food through the pylorus.

FIG. 6

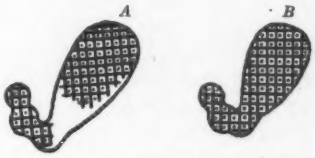


FIG. 7

FIG. 6.—Tracing of the outline of a stomach showing the separation between the walls and the food, caused by gas.

FIG. 7.—*A*. Tracing of the outline of a stomach showing a collection of gas in the pre-antral region. *B*. The same stomach after removal of gas.

The retardation of gastric discharge through the accumulation of gas in the stomach is a result which evidently might be different in man and in the cat. In the upright position of man any gas in the stomach would naturally rise to the fundus, and the food would then be in the region of active peristalsis. Furthermore, the gas might thus be readily voided by eructation. But in the prone position of man gas in the stomach may render peristaltic activities quite as ineffective as it does in the cat.

In connection with these observations there has been observed repeatedly in the pre-antral region an apparently empty space at about the position indicated in Fig. 6. Magnus[21] described a similar appearance in the stomachs of morphinized cats. He attributed the appearance to a persistent constriction of the gastric wall in this region. On two different occasions when we noted this appearance (Fig. 7, *A*) we passed a tube down the œsophagus and into the stomach, watching by means of the fluorescent screen its exact course. When the tube reached the light area, the area disappeared, and when the tube was withdrawn the shadow of the stomach presented a uniform dark appearance (Fig. 7, *B*). The light area was apparently due to gas which was carried off by the tube.

[21] Archiv f. die ges. Phys., 1908, cxxii, 254.

THE INFLUENCE OF HEAT AND COLD. The possible effects of heat and cold on the secretory and motor functions of the alimentary canal have been studied by various methods. Lüderitz[22] exposed the stomach and intestines of rabbits in a bath of normal salt solution which was gradually cooled. He saw no change in the motor activity until a temperature of 28° to 30° C. was reached. In one case very vigorous peristalsis was seen at 29° C. Below 28° the movements ceased. Oser[23] states that low temperatures close the pylorus, but that higher temperatures, up to 37° C., have no such effect. According to Müller,[24] low temperatures have a quieting, even a paralysing effect on the movements of the stomach, whereas high temperatures increase gastric peristalsis. These observations are in accord with those of Schüle,[25] who found that water at 45° left the stomach much faster than water at 0° or 28° C., but they do not seem to accord with Müller's own results that hot and cold fluids leave the stomach more slowly than fluids at body temperature.

The effect of temperature variations on secretion has been little studied. Cahn[26] reports that when he fed powdered meat with water at 15°, 1 to 1.3 p.m. of HCl was secreted during the first hour, and that with the water at 45° the acid was up to 1.8 p.m. Both these results, however, are lower than the results when temperature is normal. Micheli[27] in a study of the gastric secretion in ninety patients found that with water at 35° to 37° the secretion was greatest, at 45° to 50° least, and at 2° to 4° greater than at room temperature. A similar stimulating effect of cold water was noted by Jaworski.[28]

In such studies as are above cited the time required for the equalization of the temperature of the body and of the ingested food is especially significant, for it is probable that the temperature effects diminish as the equalization takes place. By use of maximum thermometers, Winternitz[29] observed that thirty minutes after drinking 500 c.c. of water at 5° to 7° the temperature of the gastric contents was only 0.6° C. lower than general bodily temperature. On a patient with gastric fistula Quincke[30] obtained similar results when cold water was taken, and further found that water at 40° C. reached body temperature within ten minutes. According to Quincke, hot or cold water reaches body temperature sooner than lukewarm milk. As Müller points out, the stomach is in a high degree able to bring food of widely differing temperature quickly to the tem-

[22] Virchow's Archiv, cxvi, 53.
[23] Ztschr. f. klin. Med., 1892, xx, 287.
[24] Ztschr. f. diät. und physikal. Ther., 1904, viii, 587.
[25] Ztschr. f. klin. Med., 1896, xxix, 81.
[26] Ztschr. f. klin. Med., 1887, xii, 36.
[27] Archiv ital. di clin. med., 1896. Referat, Archiv f. Verdauungs-Krankheiten, 1897, iii, 244.
[28] Deut. Archiv f. klin. Med., 1884, xxxv, 76.
[29] Physiologic Bases of Hydrotherapy, in A System of Physiologic Therapeutics, Philadelphia, 1902, ix, 41.
[30] Archiv f. exper. Path. und Pharm., 1888, xxv, 380.

perature of the body, a function doubtless dependent on the central position of the organ in the body and to the rich blood supply in its walls and in the surrounding structures.

Since the stimulating influence due to variations of temperature is present for only a comparatively short interval, the influence exerted might be correspondingly short; but the possibility of the effect outlasting for some time the period of stimulation must be considered. In the following experiments to determine the rate of discharge of hot and cold solid foods, the conditions of experimentation were quite normal. Care was taken to keep the food at the temperature stated until all had been fed.

In two cases in which the hot food was given the potato was kept in a dish surrounded by a large quantity of water at 50° to 55° C. during the period of feeding, and the animals were fed from a spoon. In the other cases the food was given by means of a syringe, and was delivered into the stomach at a temperature of approximately 60°.

The cold food was fed in a frozen condition, and reached the stomach in frozen lumps.

The following results were obtained:

HOT FOOD, 50° to 60°.			
Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	13	27	48
	10	38	45
	14	31	43
	12	31	45
	18	38	45
Average (5 cases)	13.5	33	45

COLD FOOD, FROZEN.			
Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	10	29	50
	18	43	49
	19	38	39
	14	34	44
	12	38	44
	12	25	52
Average (6 cases)	14	34.5	46.5

As the tables and the curves (Fig. 8) indicate, the only change from the normal in the rate of discharge of food, hot or cold, is a slight acceleration, but this change is so slight as to be inconsiderable. In none of the cases was there observed any notable variation from the usual peristalsis.

In a series of observations made by Mr. C. R. Metcalf in this laboratory, hot and cold applications applied from 1 to 40 minutes to the abdomen of healthy cats produced no appreciable alteration in gastric peristalsis. It continued without interruption and without evident change of rate as measured by a stop watch. On the other hand, as already reported,[21] excessive cooling of the stomach

[21] Cannon and Murphy, Annals of Surgery, 1906, xliii, 531.

and intestines, by introducing cold sterile salt solution into the abdominal cavity, may be followed by increased activity of intestinal peristalsis. But this is a procedure causing changes of temperature in the bowel too great to be produced by any external applications.

The conclusion seems justified that changes in the temperature of the food do not influence for any length of time either gastric peristalsis or the rate of discharge of solid food from the stomach.

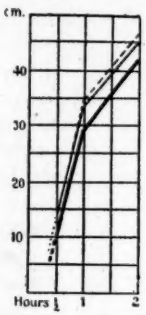


FIG. 8.—The heavy line is the normal curve for potato, the light line that for potato fed hot and the broken line that for potato fed cold.

THE INFLUENCE OF HYPERACIDITY. The earlier literature contains many conflicting statements regarding the relation between gastric motility and the secretion of HCl. Reigel,[32] Kussmaul,[33] and others express the idea that through abnormal acidity the pylorus is kept spastically closed. Similarly, Moritz[34] found in experimenting on himself that 500 c.c. of 0.95 per cent. HCl was more slowly expelled from the stomach than the same amount of water. Some of the earlier investigators regarded the acid of the gastric juice as the real stimulus to peristalsis. Later observations, however, have shown that peristalsis may be present even when the gastric contents are neutral or even alkaline.

The part played by HCl in controlling the discharge of food from the stomach and in stimulating the flow of pancreatic juice has recently been emphasized. The pylorus remains closed until an acid reaction appears in the chyme on the gastric side of the sphincter; it then opens to permit the exit of some of the chyme. Thereupon the acid chyme in the duodenum closes the sphincter, and keeps it closed until the pancreatic juice and bile, caused to flow by the presence of acid in the duodenum, have neutralized the chyme. Then and only then does the pylorus open again and let out more of the stomach contents.[35] In accordance with this explanation

[32] Ztschr. für klin. Med., 1886, xi, 17.
[33] Deut. Archiv für klin. Med., 1869, vi, 460.
[34] Loc. cit., p. 570.
[35] Cannon, Amer. Jour. Phys., 1907, xx, 293.

of the control of the pylorus, hyperacidity might very well cause a retardation of gastric discharge, but it would do so because longer time would be required for neutralization in the duodenum, and the pylorus would therefore be held closed for longer periods. It is of interest to inquire as to the effects of hyperacidity on the rate at which food leaves the stomach. To obtain evidence on this question cats were fed potato with which had been mixed a known percentage of HCl.

In determining the acidity boiled potato was mashed without the addition of water and then concentrated acid was added. Thus, for 1 per cent. acidity, 4.2 c.c. HCl with specific gravity 1.185 (*i. e.*, about 34 per cent.) was added to 100 c.c. potato, weight 140 grams. For the other percentages of acidity the corresponding fraction of this amount of acid was accurately measured and mixed with the potato. The following are the results obtained:

0.25 PER CENT. HCL.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	24	47	72
	25	35	48
	28	40	55
	34	63	64
	22	40	60
	40	52	61
Average (6 cases)	29	46	60

0.5 PER CENT. HCL.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	25	40	58
	25	41	58
	17	34	46
	20	38	49
	20	35	56
	18	30	47
Average (6 cases)	21	36.5	52

1 PER CENT. HCL.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	8	20	34
	9	16	46
	13	35	43
	9	30	72
	14	27	50
	15	19	40
Average (6 cases)	11.5	24.5	47.5

In comparing with the normal conditions these results of feeding acid food, it is fairer to use the second rather than the first half-hour of the normal curve as a standard, since at the beginning of the first half-hour digestion has not begun and no acid has yet appeared at the pylorus, while at the beginning of the second half-hour acid chyme is being discharged. As the curves (Fig. 9) and as

the tables indicate, the rate of exit is faster than normal when the potato has an acidity of 0.25 per cent., and slower than normal when it has an acidity of 1 per cent. Potato with an acidity of 0.5 per cent. is discharged during the first half-hour about as rapidly as the food is normally discharged. The difference between the outgo of the weakly acid (0.25 per cent.) and the strongly acid (1 per cent.) potato is remarkable. Note that at the end of the first half-hour there was in the intestine more than two and a half times as much, and at the end of an hour about two times as much of the weakly acid potato as of the strongly acid. The rapid exit

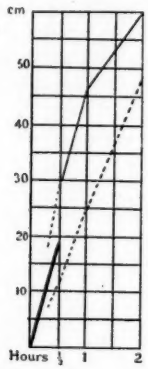


FIG. 9.—The heavy line is the curve (for the second half-hour) when potato is fed normally; the light line, when fed with 0.25 per cent. acidity (HCl); and the dotted line, when fed with 1 per cent. acidity.

was confirmed by the appearance of the stomach: in several instances the stomach was practically empty at the second observation—a condition normally never seen. The appearance of the food masses in the intestine was also characteristic, for long, continuous coils of food were regularly observed.

In all the experiments on the feeding of acid food gastric peristalsis was especially deep and rapid. The rate was usually slightly faster than six waves per minute. In one instance, at the half-hour observation, the waves passed so rapidly as to attract attention. On timing them with a stop-watch twenty waves were seen to move over the antrum in sixty-six seconds, more than eighteen per minute. This rate was so much faster than normal that the counting was repeated, with the same results. At the one-hour observation and thereafter, however, the waves passed at the usual rate.[36]

[36] Opportunity was offered to note the rate of gastric peristalsis in four kittens about six weeks old. The number of waves per minute was within the limits observed in adult animals. The rate of discharge from the stomach was likewise not remarkably different from the rate observed in adults.

THE INFLUENCE OF MASSAGE. The animals selected for observations on the effect of massage were especially docile; they were thus selected in order that the manipulation might not enrage them and thereby introduce as a complicating factor the inhibitory influence of a strong emotional state. Most of the time purring indicated a peaceful tranquillity. Massage was applied by gently kneading the abdomen, especially under the last ribs, as the animal was lying comfortably on its right side. None of the animals was massaged all the time between observations, but of six, four were massaged one-fourth of the time and two one-half of the time. In spite of this extensive manipulation there was no considerable change in the rate of gastric discharge. The average figures for the first three regular observations were 12, 25, and 36 cm., instead of the normal 10, 28, and 41.5 cm. The gastric peristaltic waves, which passed at the usual rate, were notably deep. Segmentation in the small intestine was also quite marked.

THE INFLUENCE OF IRRITATION OF THE COLON. It has recently been shown that resection of the small intestine some distance below the pylorus results in such strong and continuous contraction of the pyloric sphincter that, in spite of vigorous peristalsis, food may not begin to leave the stomach for four or five hours after feeding.[37] It is of interest to know how far along the alimentary canal such irritation may reflect its influence on the stomach. The clinical observation that inflammation of the appendix may disturb gastric digestion indicates the possibility of effects on one part of the canal from abnormal conditions in a relatively remote region. The following experiments were tried with the purpose of determining whether irritation of the colon would influence the exit of food from the stomach. In these experiments croton oil was used as an irritant. In no case did the animal show signs of discomfort from the procedure.

Five cats, a few hours after receiving a cleansing enema, were fed 25 c.c. potato and were then injected per rectum with 50 c.c. sweet oil containing 1.25 c.c. croton oil. When observed, the peristalsis of both the stomach and intestine seemed unusually vigorous. The stomach was peculiarly elongated. The rate of discharge may be judged from the following table:

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	23	39	39
	0	1	16
	16	17	33
	0	5	19
	0	12	22

The irregularities of the figures in the above table can perhaps be explained on the impossibility of clearing the colon and the

[37] Cannon and Murphy, loc. cit., p. 516.

consequent uncertainty as to the degree of dilution of the irritant by the contents of the bowel. Three of the five cases, however, showed a marked slowing of the discharge.

In order to control conditions more exactly, four cats were etherized, and, under aseptic precautions, a few drops of croton oil injected into the cecum through a small median incision in the abdominal wall. The next day they were fed the standard potato and observed. In all the animals gastric peristalsis was vigorous, but in none of them was there any discharge into the intestine during the first half-hour. In two cases gas appeared in the stomach at the end of the fourth hour, and in one of these cases so much gas accumulated that the distended stomach caused stretching of the abdominal wall and some respiratory difficulty. In another case the first food to enter the colon passed into it during one of the observations. The small mass was forced into the large intestine and pushed upward a short distance, whereupon it was violently segmented for a few moments. Thirty minutes later no more food had entered the large intestine, but a large solid coil of food, the shadow of which was about 17 cm. long, seemed to be blocked immediately before the ileocolic valve. The colon was filled with gas so that its outline was clearly visible. The following table shows the results of these experiments:

Hours after feeding	½	1	2	3	4	5	6	7	8
Aggregate length, in cm., of the food masses in the small intestine	0	0	9	25	34	34	37	45	40
	0	6	32	44	45	33	47	44	49
	0	0	6	18	21	39	41	45	51
	0	8	8	30	29	33	32	41	37
Average (4 cases)	0	3.5	13.5	29	32	35	39	43.5	44

Comparison of these figures with the figures from normal cases (Fig. 10) shows at once remarkable differences. Not only is the gastric discharge much slower when the colon is irritated, but the passage of the food through the small intestine is greatly retarded, The curve drops mainly because of the passage of material into the large intestine. Note that when the colon is irritated the curve fails to drop throughout eight hours, whereas the normal curve begins to drop at the end of two hours. Normally potato begins to appear in the colon at the end of two or three hours; under the condition of the present experiment, however, it did not appear in the colon until six or seven hours had elapsed. In all cases food was still present in the stomach at the end of seven hours, though normally the stomach is emptied of this food in about three hours.

To prove that the effects noted above were not due to the operative procedure alone, four cats were operated upon as before, and, instead of croton oil, a few drops of water were injected into the colon. In two of the cases the gastric discharge on the next

day was somewhat delayed, but not nearly so much delayed as when croton oil was injected; the other two cases were close to the normal.

The conclusion is justified that irritation of the colon can cause marked retardation in the rate of exit of food from the stomach and in the passage of food through the small intestine.

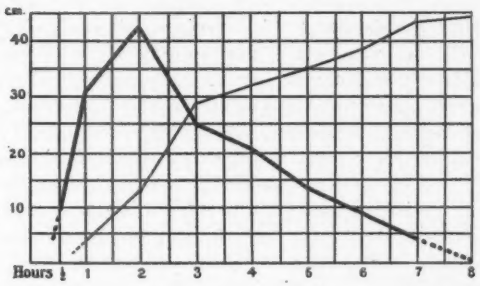


FIG. 10.—The heavy line is the normal curve for potato, the light line the curve when croton oil has been injected into the colon.

SUMMARY. If carbohydrate food is thinned by adding water, there is, within limits, very little change in the rate of exit from the stomach; but adding water to protein food tends to make the discharge more rapid. When hard particles are present in the food the rate of outgo from the stomach is notably retarded. Coarse branny food leaves the stomach slightly faster than similar foods of finer texture. The presence of gas in the stomach delays gastric discharge, an effect due to the gas preventing the walls of the stomach from exerting the normal mixing and propelling action on the food. No considerable variation from the normal rate of exit from the stomach is observed when the food is fed very hot or very cold. Food with approximately normal acidity leaves the stomach much faster than food which is hyperacid (1 per cent.), a result in harmony with other observations on the acid control of the pylorus. Feeding acid food is followed by deep and rapid peristalsis. Massage of the stomach, even when extensive, has very slight influence on the passage of food through the pylorus. Irritation of the colon (with croton oil) notably retards gastric discharge and delays the movements of food through the small intestine.

THE RELATION OF THE FOODSTUFFS TO ALIMENTARY FUNCTIONS.

By Lafayette B. Mendel,
PROFESSOR OF PHYSIOLOGICAL CHEMISTRY IN THE SHEFFIELD SCIENTIFIC SCHOOL, YALE UNIVERSITY, NEW HAVEN.

The great development of experimental research which has taken place in recent years in the study of the digestive functions has inaugurated a variety of new viewpoints in this department of physiology. In the earlier days attention was concentrated upon the processes carried out in the stomach, and the activities of the remainder of the alimentary tract failed to be drawn into consideration. The recognition of the importance of the changes which have their seat in the intestines was the outcome primarily of biochemical investigation. It is not long since we were accustomed and content solely to analyze the changes which the alimentary processes bring about in the foodstuffs—to study the effect of the digestive enzymes upon them, and to investigate the disappearance of the products from the digestive tube. Considerations such as these comprised practically the sum total of alimentation as it was then appreciated.

But instead of recounting the transformations which the foods experience in the digestive processes, instead of debating where and whether fats, carbohydrates, and proteins are completely disintegrated into simpler molecules before they are reconstructed into forms of flesh and blood, another aspect of the alimentary functions has been proposed for this discussion. The familiar query regarding the changes which digestion imparts to the foodstuffs will for once give way to a newer and reversed point of view. Let us inquire what influence the foods have on the alimentary processes. It will appear that, aside from their value as nutrients, the components of the diet exercise specific influences upon the alimentary organs. Are not such physiological agencies an appropriate theme for consideration in the field of therapeutics?

So long as the secretion of the digestive juices and the regulation of the muscular mechanisms of the gastro-intestinal tract were assumed to be under the sole dominance of a nervous apparatus, the *character* of food eaten was destined to play an insignificant part in the secretory and muscular responses. Fat and starch alike were food; and the physiologist was not inclined to discriminate between them in their action on secretion. The gush of chyme in the duodenum, like the bolus of food in the stomach, provoked a flow of digestive secretion by some obscure mechanical agency or some nervous reflex. We have become emancipated from these broad generalizations. Not all foodstuffs provoke secretion in equal degree. Not all constipation is due primarily to failure of peristalsis. The skilled

therapeutist of today can well afford to devote to the specific action of the diet the same detailed consideration which he accords to his armamentarium of drugs.

Perhaps the most fruitful and suggestive of the more recent ideas has been that of the *hormones*, or chemical excitants, which travel about the organism. Ever since Bayliss and Starling demonstrated that the flow of the pancreatic juice and the bile can be incited not only by nervous reflexes, but also by the absorption of definite chemical substances which are despatched through the blood stream to the pancreas and liver respectively, and there set up increased secretory activity, speculation has been rife as to the extent to which such chemical regulation of our functions is maintained. We have been taught to recognize the humoral paths of stimulating influences—to appreciate that active chemical agents may travel about the body from part to part, and awaken responses through the circulation as well as through the less direct nervous paths.

One of the newest of these hormones, or chemical excitants, to deserve attention in this connection has lately been brought to notice by Edkins. He has shown that the contact of certain food products with the pyloric end of the stomach gives rise to some chemical substance—a gastric hormone or secretagogue—which acts as a powerful stimulus to gastric secretion when it is introduced into the circulation. In the case of the pancreas, as is well known, the presence of acid in the duodenum awakens a speedy response: in part, this may be due to a nervous reflex; in part, probably also to the formation or liberation of a specific substance (a secretin) in the walls of the intestines, which travels directly to the glands in the blood current and provokes secretion. Now, in the stomach, acid alone is at best a slight stimulus; but dextrins, maltose and dextrose, proteoses, and, above all, extract of meat, are pronounced gastric secretagogues. They do not act from the fundus; but are absorbed in part and act from the pyloric end of the stomach and the duodenum. The first products of digestion, therefore, acting on the pyloric membrane, liberate a substance—the hormone, gastrin—which is absorbed into the blood stream and carried to all the cells of the stomach where it functions as a specific excitant of their secretory activity. Here then is furnished a definite illustration of certain food products arousing the gastric cells. We learn a dietetic method of inducing secretion in the stomach. The psychic flow which comes at the outset of the meal and has been made familiar through the researches of Pawlow is nervous in origin. It can, accordingly, be provoked, modified, or inhibited through purely nervous channels. The later and more prolonged flow of gastric juice is associated with the food derivatives. This food factor in gastric secretion is, by no means, an entirely new conception. The germ of the modern work may be found in the older observations by Schiff. But the work of Edkins and others has emphasized the specific nature of the

excitants and the fact that they act directly through absorption and not primarily on a central nervous mechanism. Bouillon or meat soup taken early in the meal soon reaches the pylorus and duodenum and incites a flow of gastric juice. The dextrins and maltose of toasted bread and zwieback perform the same function. We have thus learned an application of dietotherapy to awaken the dormant secretory glands. Abundance of secretion in the stomach in turn promises abundance of pancreatic juice and bile, because of the excitatory function of the gastric hydrochloric acid when it reaches the duodenum. The intestinal secretion is likewise said to be facilitated through the excitation by hormones.

On the other hand, fats are well known to *inhibit* gastric secretion in a striking manner. Thus, the nature of the food intake may materially modify the secretory responses of the alimentary tract and its appendages. The better appreciation and extension of the laboratory studies on the responses of the secretory glands to specific food products already give promise of useful application in the field of clinical medicine. When drugs still largely fail, a carefully selected dietetic regime may succeed in awakening or repressing organs upon the proper secretory function of which our nutritive welfare depends.

There are, furthermore, numerous and significant indirect or secondary influences, which arise from the regulatory function exercised by certain foods upon the alimentary processes. Whatever promotes an abundance of HCl in the stomach and insures its discharge through the pylorus, provokes a liberal flow of bile, and a consequent ready absorption of the digestion products of fats. Again, high or low gastric acidity markedly modifies the extent of the putrefactive changes in the bowel. The relaxation and closure of the stomach sphincters at the cardia and the pylorus are under the control (in large measure) of the reaction which prevails there. Foods which tend (like proteins) to combine with acid and diminish the "free" HCl, or (like fats) to repress acid secretion, will delay the discharge from the stomach. Whatever facilitates speedy accumulation of acid in the pyloric region tends toward an earlier emptying of the organ into the parts beyond. Newer investigations suggest that bile stimulates peristalsis in the intestines. Enough has been said to indicate how intimately and intricately the food influences may involve the ensemble of digestive phenomena.

In this connection, mention may be made of the interesting discovery, by Boldyreff, of the regurgitation of duodenal contents into the stomach under appropriate conditions. This is most likely to occur when the stomach is empty or acid secretion has been repressed to a minimum by ingestion of fats. Pancreatic juice may thus be obtained from the stomach, and the method has already been introduced into clinical usage for the diagnosis of pancreatic conditions. Migaj has suggested that strong acids in the stomach are neutralized

by such regurgitations of alkaline duodenal contents. It should be noted, finally, that even peristalsis has been brought under the alleged domination of hormones: Zuelzer claims that at the height of digestion the mucosa of the stomach yields a substance which causes energetic but natural peristalsis extending throughout the intestinal tube. This, like many of the interesting speculations which the introduction of a new idea into physiology has evolved, demands a further critical examination before it deserves to be incorporated with the more permanent acquisitions of our science.

Another and somewhat different conception of the influence of the foodstuffs upon digestive phenomena owes its prominence to the authority of Pawlow. He has taught that the secretory glands may respond in a unique way to variations in the type of foodstuffs ingested. According to him there may be a specific adaptation of the secretions, such as that of the pancreas, so that the richness of the juice in proteolytic, amylolytic, or lipolytic enzyme varies with the nature of the diet. We are taught to speak of "meat" juice, or "bread" juice, or "milk" juice, each varying noticeably in its relative digestive power. It must be admitted that an adaptive response of this sort by which more enzyme of one type or another is produced specifically as it is needed, in reply to the stimulus of the individual foodstuffs, presents a remarkable specificity of physiological function. However, the consensus of opinion at the present moment speaks against such a specific sensibility of the mucous membrane of the alimentary tract, alluring as the idea may be. One of the critics of the adaptation hypothesis (Popielski) naïvely remarks that the welfare of the organism will be better assured if we ourselves conduct the analysis of our foods than if the task is left to the digestive glands. Despite contradictory statements it appears in the case of the saliva that the texture, more than the composition of the food, is a decisive factor in awakening the secretory response.

In addition to this there is, however, a further type of claim which postulates even a more radical adaptation of secretions to the diet. The feeding of specific foods is assumed to induce the formation of desired enzymes, in some cases even when they are never found at all under usual conditions. Much more satisfactory evidence than has yet been offered will be needed to convince the unbiassed physiologist that cells can suddenly assume new roles with the ease which has been claimed—that a few days' feeding with bread or milk sugar, for example, will elicit the production of starch-digesting or sugar-inverting enzymes in secretions in which they are normally wanting. These problems of adaptation are still in the realm of the unproved. As Starling says: "There are riddles enough in physiology without conjuring up a teleological adaptation for which the experimental evidence is inadequate, the conception of the mechanism impossible, and which is not necessary for the well-being of the animal."

Examined from a still different point of view, the food factor is of

preëminent significance in relation to alimentary changes. It modifies the extent of the bacterial transformations which go on there; while Metchnikoff and his followers would have us believe that it is possible by dietetic regulation to alter decidedly the bacterial flora of the alimentary tract. The usefulness of lactic acid, sour milk, and lactic acid ferments as curative agents is nothing less than problematical at the present moment. But the very principles underlying their use emphasize the possibility of regulating alimentary changes most profoundly by the character of the food intake and its natural concomitants. The dietotherapy of infants has lately witnessed the advent of a new theory for the etiology of the alimentary intoxications (so-called) of children. Whereas, for a long time attention has primarily been directed to the dangers of the products of the intestinal flora, Finkelstein maintains most emphatically that these familiar disturbances are ofttimes nutritive (metabolic) and digestive in character, rather than more specifically bacterial. He directs attention to the food rather than the ferments in the treatment of these infantile disorders. "Food intoxication," if we may coin the expression, is by no means a novelty; nor are we prepared as yet to realize its possible consequences. A secretory function fails; fats are improperly absorbed; the fatty acids form soaps of calcium, robbing the growing organism of its supply of lime, which is wasted with the stools; acidosis intervenes. What a serious train of symptoms can here be avoided by a timely consideration of the relations of food to the alimentary processes. Drug medication is of insignificant moment amid such complications. The possibilities of the newer knowledge of the laboratory must be translated into practice and there made effective. As Professor Ewing has appropriately remarked in another connection: "The feeding of healthy man, as well as the diet of the sick, cannot be left to chance, guided by appetite, or ruled by tradition, but can be safely directed only according to the laws of digestion and metabolism."

DIET AND THE CARE OF THE BOWELS IN TYPHOID FEVER.

By M. H. Fussell, M.D.,

ASSISTANT PROFESSOR OF MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

"Lack of perception is the devil's best friend."

Typhoid fever is characterized by infection, and in the vast majority of cases by local ulceration in the ileum and the cecum. The prognosis in normal individuals depends upon the degree of the infection and the extent of the local ulceration in the intestine.

The former can be fairly well measured by the fever, the nervous symptoms, and the degree of prostration present; the latter is practically unknown. Notoriously we have as yet no efficient, specific treatment. The well-being of the patient depends upon the care he receives as to diet, bowel movements, and rest.

Diet in typhoid fever must be prescribed with two objects in view —maintenance of the nutrition and the avoidance of gastro-intestinal disturbances. It is a well-known fact that until the time of Graves, in 1840, patients with any form of fever were depleted, according to the teaching of Sydenham. Since Graves' teaching, patients with fever, notably typhoid fever, have been fed with the view of limiting the nitrogen loss to a minimum.

The diet in typhoid fever until the last fifteen years was liquid, with milk as a basis. Within the last fifteen years, however, a diet much more varied has been recommended by eminent men, notably, Shattuck, Barr, and Bushuyev. Any recommendation coming from Frederick Shattuck compels attention He cites 241 cases treated over a period of eleven years, on a most liberal diet, with great comfort to the patient and with no increase in mortality. The claims made by the advocates of a liberal diet may be tabulated as follows:

Liberal diet.	Liquid diet.
1. Satisfies the patient.	1. Disgusts the patient.
2. Provides sufficient calories.	2. Provides insufficient calories.
3. Does not produce a culture medium in the bowel.	3. Produces an excellent culture medium in the bowel.
4. Does not cause intestinal irritation.	4. Causes intestinal disturbances by curds and distention.
5. Does not increase the mortality.	
6. Does not increase perforation and hemorrhage, but rather diminishes them.	6. Increases perforation and hemorrhage, because of curds.

Taking these claims up seratim:

1. The comfort of the patient is unquestionably greater when he is given food other than milk and water. Three or four weeks of an absolute milk diet is most trying to the patient if he be not very ill, and is perhaps still more trying during convalesence. No one who has seen the change in mental attitude of a patient on milk when he is ordered milk in the form of junket, is given ice cream, or thin cornstarch, egg, or other suitable diet, can doubt its good effect, mentally, at least. Those who are in the habit of giving the diet suggested by Shattuck claim still greater improvement of the moral.

2. The usual amount of milk given in the course of a liquid diet, maintained at St. Timothy's Hospital, Roxborough, Philadelphia, varied from 48 to 72 ounces in twenty-four hours, in each of 514 cases, and this I think agrees well with the usual amount given. According to Chittenden, 2 quarts of milk supply 1300 calories. To supply 2700 or 2800 calories, which in health is Chittenden's necessary amount, each patient should receive 4 quarts of milk. Our

patients had no more than 2½ quarts, or 1625 calories. We must acknowledge that, theoretically at least, these 514 patients were decidedly underfed. The result of these cases, however, which agrees fairly closely with other groups of cases, would go to show, that however much more comfort can be obtained from a fuller diet, so far as recovery is concerned, it is not a necessity. A large number of metabolism experiments made by Puritz,[1] in 1890 to 1892, in which typhoid fever patients were fed on meat, bread, milk, eggs, tea, coffee, and port wine, shows that from 79 to 82 per cent. of the nitrogen consumed was assimilated. These experiments go to strengthen the position of the adherents of a more liberal diet, for if 80 per cent. of the nitrogen consumed can be assimilated, certainly it is common sense to give it to the patient, provided it does not otherwise harm him.

3. The claim that milk as compared with solid foods causes a good culture medium in the bowel, is not well founded. Certainly the entire intestinal canal is swarming with all sorts of bacteria. It matters not, it seems to me, whether the food be taken as milk or more solid food, that which remains must provide the culture medium. There is no more refuse in the bowels after a diet of milk than after other diets. In many autopsies, made by myself in St. Timothy's Hopsital, I have failed to find the lower bowel overfull, or containing a large number of curds, though the patients had consumed nothing but milk during the course of the illness.

4. Does milk cause more or less intestinal irritation than the same quantity of other food? One of the chief objections to a liberal diet, in the minds of those who use a liquid diet, is the fear that the solid food will leave so much residue in the intestine that the ever-present ulcerations will be irritated, and the intestinal accidents, perforation, and hemorrhage increased. It must be remembered, in this connection, that milk on entering the stomach at once becomes a solid food, although easily digested.

Statistics published by Kinnicutt go to show that perforation and hemorrhage are slightly less in patients on a free than in those on a milk diet, but aside from this, which will be considered later, the tympany and diarrhœa which some writers attribute to milk diet are more in the imagination than in reality. When tympany does occur with a milk diet, when diarrhœa is excessive, the milk may be withdrawn; it may be substituted by peptonized milk, junket, or albumen water. In the series of cases from St. Timothy's Hospital diarrhœa occurred 113 times, but it was rarely excessive; when more than four stools occurred daily, it was considered diarrhœa. It would appear, therefore, and this has been my personal experience, that milk diet does not, as a rule, cause undue distention or diarrhœa; when it does the diet may be controlled, so that the

[1] Quoted by Atwater and Langworthy.

difficulty may be obviated. We may well judge the effect of a liberal diet and a liquid diet upon mortality, perforation, hemorrhage, and relapse, by a collection of a large number of cases. It must be remembered, however, that statistics alone are not to be depended upon for information, however great their help. To help decide this question, Kinnicutt has published some remarkable statistics collected from different hospitals. I have another series to submit from St. Timothy's Hospital.

KINNICUTT'S TABLES. Free Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
733	48	15.2	35	4.77	10	1.36	60	9.47
Basis of	315 cases		733 cases		733 cases		633 cases	

Liquid Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
4654	507	10.89	411	8.83	111	2.38	491	10.55

ST. TIMOTHY'S HOSPITAL TABLES. Liquid Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
514	43	8.36	54	10.50	21	4.00	69	13.42

COMBINED TABLES. Liquid Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
5168	550	10.64	465	8.99	132	2.74	560	10.83

Dr. J. C. Wilson[2] has published a series of 1904 cases of typhoid fever treated by the method of Brand with a mortality of 7.5 per cent. No mention is made in the article as to the diet, and certainly the cases were not reported with that end in view. As all the cases were treated in Philadelphia, it is presumed that the cases were given a liquid diet. The series shows, however, that the diet alone is not the only factor which affects the mortality.

These tables, so far as they are of value, show the following. In the first place the mortality is somewhat less in the series on a liberal diet. In 633 cases fed on liberal diet, there was a mortality of 9.47 per cent. In 5168 cases on milk diet, the mortality was 10.83 per cent.—a difference of a little over 1.75 per cent. in favor of a free diet. Relapse was present 550 times, or 10.64 per cent., in liquid diet as against 15.2 per cent. in free diet. Hemorrhage was present in 8.99 per cent. as against 4.77 per cent. in liberal diet. Perforation was present in 2.74 per cent. as against 1.36 per cent. in liberal diet. These differences are certainly negligible. For instance, of my own cases, which are all classed under liquid diet, and were treated other-

[2] Phila. Med. Jour., March 3, 1900.

wise entirely differently, several died in a few hours after their admission. The diet had nothing to do with their demise. In one year at St. Timothy's Hospital there were 12 deaths. Every death was due to perforation; certainly two patients were admitted with a perforation, and one died of this accident one week after the patient was on soft diet. If these cases were excluded, the statistics would vary decidedly. In that particular year there would have been the difference between 9 and 12 per cent.

We, who have seen the stools of normal adults and children after eating immoderately, and know how they are filled with curds if given large quantities of milk, undigested particles of meat, etc., can realize the foolhardiness of using any diet in typhoid fever except under strict watchfulness. Any physician of experience will be certain, without statistics, that gross errors in diet do cause both relapses and intestinal accidents in typhoid fever. Two such cases come to mind as I write; they could be multiplied many times. One, a case in St. Timothy's Hospital, which was convalescent without any symptoms of any kind, at the end of two weeks, while on soft diet developed, a sudden rise in temperature, and in two days after, a perforation. At the operation and subsequent autopsy the intestine was found fairly well filled with fecal matter, which had perforated an ulcer. It would appear that this particular perforation occurred because of irritation of an unhealed ulcer by a large fecal mass. A second case, a girl, aged twelve years, had passed through a severe attack of typhoid fever. She had a normal temperature for ten days. The family was told that her milk might be increased. They at once gave her six quarts in twenty-four hours, with an immediate result of a severe relapse. Certainly, it appears that both of these results were due to excess in diet, one of milk, the other of a liberal diet.

If hemorrhage occurs, all food must be withdrawn. Water may be given in small quantities; in twenty-four hours albumen water, then peptonized milk. After one week the usual diet may be resumed. If perforation occurs, there is no question of diet. The case must at once be handed to the surgeon for operation. Thromboses are said to be much less frequent in cases well fed than in cases fed on liquid diet. There are no statistics known to me to prove this point. My own cases cannot be used for this. I believe there is such a thing as fever of malnutrition following a liquid diet. Patients may run a temperature of one or two degrees above normal for days and weeks after a normal temperature. In these cases I think food must be given. It is claimed that they do not occur with a full diet.

Shattuck, in discussing Kinnicutt's paper in 1906, said: "For more than ten years I have been feeding my patients according to their digestive power, rather than according to the name of the disease, avoiding such articles of diet as can leave an irritating residue. I am convinced that comfort is promoted and convalesence shortened."

Bushuyev treated 398 patients in the Military Hospital in 1895 to 1897. He[3] is most enthusiastic, saying: "Under this regimen the general condition of the patients is incomparably better than when kept upon an exclusively liquid diet. The common complaints are scarcely ever heard. At breakfast, dinner, and supper the patients are uncommonly wide awake. Even those who are very ill sit up in bed, beg for food, and eat with much satisfaction; only a few have to be fed by nurses. If one observes the patients at meal times he wholly forgets that these individuals are seriously ill with temperature above 39° C. During the first hours in the ward the patient lies in a motionless condition, failing to answer questions and refusing food. But if one succeeds in some way or another in persuading him to eat a bit of meat or cutlet, or an egg, he immediately begins to show some interest in his surroundings."

These are certainly commendatory sentences. In this country we would doubt the advisability of patients sitting up in bed with typhoid fever, even though they were able. Bushuyev treated 318 cases on the diet described, with a mortality of 8.2 per cent. as compared with a mortality of 12.4 per cent. in 2887 patients in the same hospital during the ten preceding years.

If one compares the diet list of Shattuck with that of Bushuyev, both of which are appended, it is easy to choose between the two. Bushuyev gives no directions as to methods of feeding, nor preparations of foods. Shattuck's is the more rational.

My advice as to diet for typhoid fever cases is to study each patient well, and feed him according to his needs. First, put the patient on a liquid diet, with milk and egg albumen as a basis. Increase the diet up to a limit of 2500 calories, by the addition of starches, minced chicken, or meat, chopped soft parts of oysters, soups, after the diet of Shattuck. Keep a careful watch upon the stools and on the digestive functions, to see that neither undigested curds or other particles pass, and that tympany and diarrhœa and constipation do not occur. The presence of particles in the stool, whether milk curds or other matter, is at once to be taken as an indication to remove the offending article of food from the diet list, or change its character. If diarrhœa or tympany continue, stop all food and begin again with digested milk, eggs, etc.

To paraphrase Kinnicutt, the employment of a fixed diet in all cases of typhoid fever would be as irrational as a decided limitation of diet in many cases of typhoid fever.

The care of the bowel movements is akin to that of the diet. The keynote must be the avoidance of routine. It would appear certainly unscientific to give salines constantly to a patient already suffering from an irritated and ulcerated intestinal mucous membrane. On the other hand, it is irrational to allow a patient to go several days

[3] I quote from a translation by Kinnicutt.

without a bowel movement. While the first practice irritates the bowels by undue action, retention of fecal matter may allow the patient to absorb deleterious substances, or the retained fecal matter may irritate the ulcers by its presence. The safe course is to give the patient divided doses of calomel, if seen in the first week, in order to remove any irritating substance that may remain in the bowel. After that, a daily evacuation must be secured by a glycerin or a simple enema, if there is a tendency to constipation. If diarrhœa occurs, food should be stopped or regulated. If it then persists, a mixture of salol and bismuth should be given, or an opiate if excessive.

To conclude: Clinical and autopsy evidence proves that a regulated milk diet does not irritate the bowel, neither does a diet of milk re-inforced by carefully selected carbohydrate and nitrogenous diet other than milk. Common sense must be applied to both, and both may be used, but in semiconscious and unconscious patients the diet is perforce liquid. The excessive excavations should be controlled if diarrhœa is present. One bowel movement should be obtained by daily enema if there is constipation.

Shattuck's dietary in the Massachusetts General Hospital consists of:

1. Milk, hot or cold, with or without salt, diluted with lime water, soda water, Apollinaris, Vichy; peptogenic and peptonized milk; cream and water; milk with white of egg; slip buttermilk, koumyss, matzoon, milk whey, milk with tea, coffee, cocoa.

2. Soups: Beef, veal, chicken, tomato, potato, oyster, mutton, pea, bean, squash; carefully strained and thickened with rice (powdered), arrowroot, flour, milk or cream, egg, barley.

3. Horlick's food, Mellin's food, malted milk, carnipeptone, bovinine, somatose.

4. Beef juice.

5. Gruels: Strained cornmeal, crackers, flour, barley water, toast water, albumen water with lemon juice.

6. Ice cream.

7. Eggs: Soft boiled or raw; eggnog.

8. Finely minced lean meat, scraped beef. The soft part of raw oysters. Soft crackers with milk or broth. Soft puddings without raisins. Soft toast without crust. Blanc mange, wine jelly, apple sauce, and macaroni.

The total number of cases treated by Shattuck on this principle is 241, extended over the period from 1893 to 1904.

Bushuyev's daily menu in the Kief Military Hospital, Russia, is as follows:

7 A.M. Tea with roll.

8 A.M. 400 c.c. of soft (liquid) oatmeal, barley, or wheat porridge, with butter.

9 A.M. One or two boiled eggs, soft or hard, as the patient desires.

10 to 11 A.M. A glass (200 to 220 c.c.) of milk with a roll, one-half a cutlet, and a bit of boiled meat (160 to 168 grams).

12 to 12.30 P.M. A plate (220 c.c.) of chicken soup or a bowl of ordinary soup, sometimes with a bit of chicken from the soup, and a small cup of "kisel" (a sort of sour jelly); rarely, a little preserved fruit.

3 P.M. Tea with roll.

6 P.M. A cup of chicken or beef soup; semolina pudding or milk, a bit of chicken.

8 P.M. Milk with roll.

During the night: Coffee or tea with milk, two or four times; coffee with cognac.

In addition, the patients receive from one to three ounces of wine in the morning, and every two hours half an ounce of Stokes' mixture.

THE ETIOLOGY OF LOOSE BOWEL MOVEMENTS.

By A. D. Blackader, B.A., M.D.,

PROFESSOR OF PHARMACOLOGY AND THERAPEUTICS IN MC GILL UNIVERSITY, MONTREAL; PHYSICIAN TO THE MONTREAL GENERAL HOSPITAL.

Before considering from a clinical standpoint the etiology of loose movements of the bowels, a brief statement of the physiological facts which have a bearing on the condition may be of service. In the intestinal tract we have to deal with a canal which is lined with various forms of glandular epithelium, and which has to perform the important functions of digestion, absorption, and, finally, elimination. Its activities are presided over by a sensitive nervous system energized from, and sympathetic with, the nervous centres in the cerebrum and cord. For its nutrition it is dependent upon the general blood and lymph stream, and is an early sufferer when these are at fault. Moreover, it is in direct communication with the external air, and becomes a culture ground for bacteria, some harmless, possibly even beneficial, while others are potentially dangerous and on occasions may be the cause of much disturbance and may even bring death to the whole organism. These functions and conditions are all mutually complimentary, so that disorder in one promptly leads to disturbance in the others.

As one of the by-results of the effective co-working of these functions, the majority of individuals secure one daily evacuation of fecal material of a soft-solid consistence, from which nutritive material has been almost entirely abstracted. In a few, evacuations may number two or three per day, and in another small group of individuals the frequency of the evacuations may be reduced to one every second

or third day. Both these conditions verge on the pathological, but so long as the general health in no way suffers and the movements are moderately firm in consistence, they can scarcely be said to demand interference on the part of the physician. An alteration in the consistence of the evacuations, however, must be regarded as more important. The contents of the small bowel are normally fluid and pass in from four to six hours from the stomach to the cecum. Considerable inspissation takes place in the ascending colon, and the now slowly moving contents of the transverse and descending colon acquire, under normal conditions, a distinctly firm consistence. The presence of an undue amount of water in the evacuations must, therefore, be regarded as indicative of some disordered function, the importance of which will vary according to the underlying cause, and to the systemic effects produced.

Looseness of the bowels, as it is popularly termed, may be dependent primarily upon: (1) Excessive propulsive peristaltic action; (2) an abnormal increase in the amount of secretion from the mucous membrane and secreting glands of the canal; and (3) defective absorption. These may work in a vicious circle. Increased peristalsis prevents absorption, and is often associated with increased secretion; increased secretion stimulates peristalsis and interferes with absorption; and defective absorption stimulates peristalsis, and is generally associated with some fault in secretion.

Although some physiologists regard true peristalsis as, in part, of myogenic origin, experiments indicate that it is, for the greater part at least, reflex in character, and that the stimuli which evoke it arise almost entirely from the bowel contents; these stimuli are directed and energized by the plexuses of Meissner and Auerbach, but are modified and controlled by impulses from the central nervous system through the vagus and splanchnic nerves.

Such stimuli may be the result of mechanical or of chemical irritation. The bowel contents act mechanically as stimulants to peristalsis by their bulk and by the coarseness and indigestibility of their particles. Chemical irritation arises for the most part from acidity or acridity in the contents; such acidity or acridity may develop as a result: (1) Of irritants introduced with the food; (2) from irritants the product of faulty digestion; or (3) from irritants developed from abnormal fermentation or other undue bacterial activity in the intestines.

The action of these stimuli will be increased by any abnormal irritability of the bowel wall including any inflammatory condition of the mucosa, or any excess of irritability in muscle fibers or in the nervous centres. Still further the influence of mental fatigue and mental emotions over the activities of both stomach and intestines is well recognized, especially in the hysterical and neurasthenic.

Secondly, undue fluidity of the evacuations may have as their primary cause excessive intestinal secretion. The secretions from the

stomach, the liver, the pancreas, and from the intestinal glands proper, are, as we all know, very abundant, but in normal conditions much of their watery, and some of their solid, constituents are re-absorbed into the general circulation. Should absorption be impaired and true peristalsis be unduly active, this re-absorption takes place imperfectly and fluid movements will result.

All local irritants also to some extent stimulate secretion; some bacterial toxins appear to act in this way, producing excessive secretion of watery fluid, followed by copious evacuations, as for example, the rice water discharges of cholera. The presence of certain salines in the bowel contents also stimulates secretion and by osmotic action checks absorption. It must not be forgotten also that the large epithelial cells of the colon act as eliminating organs and that the toxins eliminated by them not infrequently stimulate peristalsis.

Loose evacuations may be primarily dependent upon defective absorption. Defective absorption may in some measure be due to imperfect digestion and preparation of the food for absorption. More important, however, is the interference due to conditions which interfere with the vital activity of the columnar cells lining the mucosa, to degenerative changes affecting the permeability of the walls of the intestinal blood vessels, and to any stasis of the portal or lymphatic circulation, all of which conditions may have an important place in the etiology of loose movements.

Closely associated with these conditions, sometimes as a cause and sometimes as a sequence, is an abnormal development of bacteria in the intestinal tract. Although the infant is reported to come into this world with sterile intestines, contamination of them quickly occurs, and the canal of an infant a few days old is said to contain a numerous flora. In the adult stomach numerous microörganisms, derived in part from the ingested food and in part from the air, are found. The intestines also, normally, contain many forms of bacteria to which the system has grown accustomed, and which are said to favor the natural digestive changes taking place in the intestine. In the small intestine no one form appears to be constantly present, but the flora varies from time to time according to the character of the diet. Most of the bacteria found in this portion of the canal appear to act on the carbohydrates in preference to the proteids. In the large intestine Bacillus coli is constantly present, and although ordinarily harmless, may at times vary much in virulence and in the toxicity of its products. To what extent these normal bacteria may induce disorder has not been fully determined. Their undue growth is held in check in the stomach by the presence of free acid and by the process of digestion proceeding with due rapidity; in the intestine, by the biliary acids, by the acids resulting from the decomposition of fats, and possibly by other digestive secretions, and also by the normal rapidity of intestinal

digestion and absorption. Disturbances of digestion and motility may antagonize or inhibit these preventive measures and either favor an excessive growth of such bacteria as may exist in the canal, or bring about a marked increase in their virulence. Under these conditions irritative conditions of the mucosa will surely develop.

With the introduction of new forms, and especially with the introduction of pathogenic bacteria, much disturbance often arises; but not always, for such may be either destroyed or rendered inert by the same natural defences which check the undue development of the normal flora. There is also considerable experimental and clinical evidence to prove that the normal bacteria of the intestine are distinctly useful in protecting the individual against new invaders.

In considering the etiology of loose evacuations from a clinical standpoint, we note first that loose evacuations may be occasioned by the mechanically irritating character of the food. It is generally recognized that coarse foods, fruits and vegetables containing much cellulose, berries and small fruits containing numerous seeds, by mechanical irritation of the mucosa stimulate peristalsis, and may, under conditions of increased susceptibility, lead to loose movements. The ingestion also of an unduly large amount of fatty food, such as oil and butter, may lead to increased peristalsis with the appearance of the unabsorbed fat in the motions. More important, however, as an etiological factor in the production of loose movements, is a faulty performance of the gastric functions. Both defective secretion and defective motility favor fermentation in the gastric contents and lead to the development of irritating organic acids and gases, and also to a great increase in bacterial growth. Hypersecretion with marked hyperacidity of the gastric contents may, by the discharge of extremely acid chyme into the duodenum, neutralize the normal alkalinity of the contents of the small intestine, and by so doing inhibit the action of the pancreatic enzymes, irritate the intestinal mucosa, and interfere with absorption.

The exact effect on peristalsis of intestinal indigestion due to deficiency in the secretion of the bile and pancreatic juice cannot be stated positively. Clinical and experimental studies have given contradictory results in the experience of different observers. Under normal conditions both appear to be secreted in quantities much in excess of actual requirements. A marked deficiency in the pancreatic secretion undoubtedly interferes with the splitting up of the fatty elements of the food, and consequently with their absorption and assimilation; it also interferes with the necessary changes which the proteid elements have to undergo to fit them for absorption. Any deficiency in the bile intensifies this condition as regards the fats. Experiments show that when the bile is absent, from 60 to 80 per cent. of the fats escape absorption, but digestion of the carbohydrates and proteids is only slightly interfered with. Of the succus entericus we know little.

In all conditions of impaired functional activity of the digestive secretions the development of a large bacterial flora promptly occurs, in part owing to the tardiness of the digestive process, and in part to the increased amount of nutritive material escaping unabsorbed into the lower portions of the intestine and affording a richer culture medium. Such development generally leads to loose movements associated with a varying group of other symptoms, but in the present state of our knowledge, we cannot definitely state that any one form of bacterial intoxication is the specific and only cause of any one particular group of symptoms.

In these conditions of impaired intestinal digestion as the result, in part, of defective absorption, in part, of the irritating changes taking place in the bowel contents reacting on a mucosa rendered unduly sensitive, and in part, of the direct action of bacteria or their toxins, peristalsis is generally unduly stimulated and the semifluid contents are hurried along without due inpissation.

Again, the clinical investigator must always bear in mind that in some individuals the intrinsic nervous mechanism of the intestines is particularly susceptible to impulses received from the large nervous centres in the cord and cerebrum. Emotion, worry, dread of any kind, and especially sudden fright, may evoke imperative loose movements: in some by increasing motility, in others by leading to profuse watery secretion, which by some physiologists has been likened to the sudden increase in the amount of saliva secreted under nervous influences. Chilling of the lower extremities may act in the same way. The morning diarrhœa of neurasthenic individuals is due apparently to some temporary irritable condition of the lower bowel, inducing one or two early morning evacuations which empty the lower bowel, and then do not recur for the day. Diarrhœas attributable to this excessive nervous irritability are not infrequently observed in Graves' disease, in hysteria, and in neurasthenia.

Loose evacuations are occasionally due to a vicarious elimination of toxic products accumulating in the blood. It has been recently shown that the toxin of Bacillus dysenteriæ injected hypodermically is eliminated by the large epithelial cells of the colon and is a potent cause of the diarrhœa. A diarrhœa of elimination may occur in many infectious fevers, in septicemia, in pneumonia, and in malaria. The diarrhœa of Bright's disease is well known to all. In gouty attacks any checking of the arthritic symptoms may induce diarrhœa. Sudden alteration of temperature may also induce diarrhœa, due in some measure to a sudden checking of the perspiration, and in part to reflex nervous influences.

Lastly, we may meet with loose evacuations due to disturbances of the blood or lymph circulations, such as the congestion associated with a failing right ventricle, or with a blocked thoracic duct.

CHRONIC CONSTIPATION CLINICALLY CONSIDERED.[1]

By Louis M. Gompertz, M.D.,

CLINICAL INSTRUCTOR IN GASTRO-INTESTINAL DISEASES IN YALE UNIVERSITY; GASTRO-ENTEROLOGIST TO THE HOSPITAL OF ST. RAPHAEL, NEW HAVEN.

There is no single condition that is more often the cause of semi-invalidism than chronic constipation; no organ in the body is exempt from its far-reaching influence, although the effects produced are mainly concentrated on the nervous system and demonstrated in certain disturbances of general metabolism. No attempt will be made to enumerate the manifold symptoms due to constipation; but a few of the changes which take place and are observed clinically may be worthy of emphasis. These may present themselves as purely local conditions or as systemic or reflex disturbances. Patients often come complaining of vague digestive troubles, such as pain, pressure, or discomfort in some particular region or over the entire abdomen. On careful questioning the clinician soon learns that these symptoms entirely disappear after a thorough movement of the bowels has taken place. In this class of cases the nervous phenomena are so pronounced that, despite the existence of organic changes, which may perhaps be overlooked, these patients are often called neurasthenics; and they drift from one physician to another, soon falling an easy prey to the quack and patent medicine man.

What is meant by chronic constipation? A well-known dictionary defines constipation as "a state of the bowels in which the evacuations do not take place as frequently as usual, or are inordinately hard and expelled with difficulty." The study of a number of text-books, with a view to determining the factor involved in the first part of this definition, namely, the normal period after which the food residues should be evacuated in a healthy individual, failed to disclose any satisfactory data. The writer, therefore, concluded to obtain first-hand evidence on this important physiological problem, in the following way:

Three capsules each containing 10 grains of lampblack were given to various individuals at different times during the day, and the number of hours required for the appearance of the black in the stools was noted. The results of observations on thirty individuals indicate an interval varying from twelve to fifteen hours until the lampblack could be identified in the feces. The following table is appended to show a few of the results obtained.:

[1] Read at a meeting of the American Therapeutic Society, New Haven, May 7, 1909.

Sex.	Age.	Occupation.	Habits.	Capsules taken.	Black in stool.	Time. Hours.
Male	36	Physician	Active	8.00 A.M.	7.00 P.M.	11
Male	32	Merchant	Active	8.00 A.M.	9.00 P.M.	13
Male	28	Student	Active	6.00 P.M.	9.00 A.M.	15
Male	27	Student	Active	7.00 P.M.	10.30 A.M.	15½
Male	24	Student	Active	9.00 P.M.	10.30 A.M.	13½
Male	23	Student	Active	7.30 P.M.	9.30 A.M.	14
Male	24	Student	Active	7.00 P.M.	8.30 A.M.	14½
Male	22	Student	Active	7.00 A.M.	8.00 P.M.	13
Male	24	Student	Active	8.00 A.M.	9.30 P.M.	13½
Male	24	Student	Active	10.00 P.M.	12.30 A.M.	14½

All of the persons examined were accustomed to have a movement of the bowels in the morning. The experiment shows that, without exception, lampblack taken with the evening meal had reached the end of the alimentary tract by the following morning. Black taken with breakfast usually reappeared the following morning; but in individuals accustomed to defecate later in the day, it not infrequently made its appearance in the evening hours. From this it becomes evident that the common practice of civilized man to discharge the feces in the morning is based on the physiological experience that the food and digestives residues of the previous day's intake have had abundant opportunity to traverse the alimentary tract.

Three clinical varieties of constipation are familiarily described, namely, the atonic, the spastic, and the fragmentary. I shall discuss only the first two of these, believing, as I do, that the fragmentary variety is merely a manifestation of the spastic form.

The atonic type is the first stage of chronic constipation, and is due to an atony of the muscles of the large intestine, usually induced by the use of purgatives. There is no doubt that lack of tone of the intestinal musculature plays a prominent part in this form of constipation, but it has been pointed out by Schmidt and Strassburger that a very complete digestion and absorption of the food in the intestine is also a prominent factor, in that it tends to deprive the alimentary residues of their usual bulk and semisolid texture. In the atonic form of constipation the stool is hard and dry; the caliber is usually very large. No specific symptoms are in evidence. There is no pain or flatulence. After this manifestation has existed for a number of years, there results a catarrhal condition due to the irritating effect of the impacted fecal masses upon the intestinal mucosa.

The conditions here outlined are frequently succeeded by a hypertonicity of the musculature of the intestine, giving rise to the variety known as spastic constipation. This form is usually accompanied by flatulence and a catarrhal condition of the intestine. Spasm of the transverse colon occurs, causing a retention of firm masses of feces, with resulting fermentation. Colic is a frequent symptom, and in severe cases large amounts of mucus are passed. In spastic consti-

pation the rectum is usually empty, while in the atonic variety the opposite condition prevails. On examination the transverse colon can be palpated, rolling beneath the fingers as a hard cord. Patients often complain of pain in this situation. In contradistinction to the atonic form, the stool is of small caliber, and is at times even ribbon-shaped. In many cases small, hard, dry segments are passed, initiating the so-called "fragmentary type." Oftentimes the organism attempts to get rid of the retained feces by causing violent contractions of the intestines, with a resulting mucous colic or diarrhœa. This may alternate with constipation.

It may not be easy to understand how temporary loss of memory, albuminuria, convulsions, numerous skin affections, and many other functional and organic changes can be caused by chronic constipation, but that they do occur is the common experience of the observing clinician. Many believe that the absorption of toxic products, the formation of which is facilitated by the fecal retention, is the cause of these numerous symptoms. This theory, however, is by no means universally accepted. Numerous cases are cited in which healthy individuals rarely have a movement of the bowels more than once in forty-eight hours, or even longer, yet suffer no unpleasant symptoms. While this may be true, it is an accepted clinical assumption that oftentimes an intestinal absorption of toxic compounds, formed in the gastro-enteric tract, originate changes in the liver, kidneys, brain, and bloodvessels.

In a number of patients whose only obvious ailment was chronic constipation, I have repeatedly observed an excretion of albumin, which promptly disappeared as soon as regular movements of the bowels were initiated. A few cases have come under my observation in which temporary disturbance of psychic functions has occurred, due, no doubt, to habitual constipation. It is a common experience to see various skin affections disappear after proper diet and a daily movement of the bowels. It is not unreasonable to suppose that chronic constipation is often the cause of arteriosclerosis. The constant absorption of gastro-intestinal toxins introduces an irritant for the bloodvessels, and thereby may cause an abnormal growth of fibrous tissue. I believe, therefore, that the auto-intoxication theory is one that deserves a great deal of consideration. In localities where malaria is prevalent this disease is often regarded as responsible for the malaise, headache, giddiness, chills, fever, etc., in patients whose only ailment is that affliction of modern civilization, chronic constipation.

For the successful treatment of constipation it is exceedingly important to ascertain the factors producing the condition. A few of the most frequent causes of constipation are the following: (1) Dietetic errors, a too perfect absorption of food substances; (2) gastrogenic constipation, increased acidity of the gastric juice; (3) irregular habits, the outcome of carelessness; (4) insufficient

exercise, sedentary habits, and obesity; (5) nervous influences, hysteria, diseases of the nervous system; (6) drugs; and (7) local obstructions, stenosis of the intestinal tract due to neoplasms, etc.

Hyperacidity of the gastric juice is an important etiological factor, resulting in the so-called cases of gastrogenic constipation. In a series of 100 cases of habitual constipation, taken from my records, without any attempt at selection, 87 cases showed an increased acidity, after an Ewald-Boas test breakfast. On the other hand, in diarrhœa, an analysis of the stomach contents regularly showed a decreased acidity. Constipation due to hyperacidity has been explained by Strassburger, who believes that the strong disinfectant influence of the increased hydrochloric acid causes a more perfect disintegration and sterilization of the food, thereby diminishing the number and retarding the growth of intestinal bacteria. Thus, the putrefactive changes in the bowels are decreased to such an extent that the natural irritation is removed, causing a lessened peristalsis, with resulting constipation. The microscopic examination of the excrement usually discloses the residue to be mostly detritus.

This brings me to the consideration of diet as a cause of constipation. The unusual dryness of the stools has been explained by a too perfect absorption of the foodstuffs and water. The chemical investigations of Schmidt confirm this condition, as he found that the fat, cellulose, and even nitrogen content were markedly diminished.

Studies made in the laboratory of Prof. Lafayette B. Mendel, of Yale University, have shown that whereas ordinary carbohydrates (starches and sugars) are very perfectly utilized in the alimentary tract of man, a considerable number of unusual carbohydrates, such as occur in many seaweeds, etc., are not attacked by the digestive enzymes. Experimenting with agar agar, for example, he found that the greater part was excreted in the feces unchanged. As agar agar absorbs water readily and retains it, and as it is able to resist the action of intestinal bacteria, as well as the enzymes, its value in the treatment of chronic constipation was suggested to Prof. Mendel. Such compounds might give texture to the stools without introducing any objectionable products of decomposition. In Mendel's experiments in which agar agar was fed, it could be recovered readily from the stools. The effect on the total mass of feces passed is noteworthy. The agar easily retains water in the alimentary residue and prevents the formation of the scybalous masses so characteristic of spastic constipation. It was this property of giving a soft consistency to the rectal contents, together with the resistance of the agar to bacterial decomposition, with the production of gases or other noxious products, which led to the suggestion of its use in the dietotherapy of constipation.

In order to test Mendel's theory clinically, I have systematically given agar agar to patients suffering from chronic constipation. Thirty such patients have been under observation. These were

from all walks of life; their ages varied from fifteen to eighty-three years. The majority of them were unable to have a movement of the bowels without the aid of medicine. The agar was taken morning and evening, beginning with 15-gram doses, and in most cases was eaten with milk or cream, the same as a modern breakfast food. In all but two of the cases, regular movements of the bowels began after the agar had been taken for from one to three days. In the successful cases after regular movements of the bowels had commenced, the agar was gradually reduced. The duration of treatment varied from one to three months. The following will serve as an illustration:

A clerk, aged thirty-one years, consulted me for the first time on March 8, 1909. His most prominent symptoms were headaches, accompanied by temporary lapses of memory, when bowel movements failed to occur. The clinical diagnosis was spastic constipation. The patient was given agar agar, and after three days' treatment regular movements of the bowels began. As improvement continued the amount of agar agar was gradually reduced to 5 grams per day, which is now sufficient for daily evacuations. During the course of treatment, he was unable to procure the agar for four days, but in this interval normal bowel movements occurred for two days.

Another case illustrates the results achieved in the atonic variety The patient, aged eighty-three years, has not had a normal movement of the bowels in twenty-five years without the use of drugs. He has been taking agar agar for six weeks, beginning with 15-gram doses twice daily. After three days' treatment regular bowel movements occurred. The dose has been gradually reduced to 10 grams daily. A further reduction of the amount of agar in this case failed to produce results.

The effect of the agar agar on the stool in the illustrated cases confirms Prof. Mendel's theory. In all cases there was a pronounced increase in the volume of feces passed, and instead of the small, hard, dry scybalous masses, the evacuations were well formed and of a dough-like consistency. In a few patients the agar in 15-gram doses produced a diarrhœal stool. When the dose was reduced normal evacuations returned. Clinical experience with agar agar tends to show that the rational treatment of constipation resulting from a too complete absorption of food substances and water is one of diet rather than of drugs. It is well known that the management of constipation by the use of drugs affords only temporary relief, and many times induces an impaired tonicity of the intestinal musculature, thereby aggravating the condition. The important factor in dietary treatment is to bring about a condition in which a perfect selection of foods would avoid the hardness and dryness of the feces in the intestines.

The foods to be selected and the combinations necessary for

individual requirements are so varying and manifold that usually only a long series of experiments, differing in each case, can possibly bring about ideal results. Here by the use of agar agar, in itself a simple carbohydrate, taken from seaweed, clinical as well as laboratory experiments have demonstrated the possibility of transforming the hard and dry solidity of the feces into a condition of softer consistency, so that the intestinal contents develop their own agency necessary to produce a healthy, normal evacuation.

My experiments may not be extensive enough to arrive at final conclusions, but the fact that the results obtained are consistent and continuous, unlike the diminishing and aggravating effects of drug treatment, leads us to look for great possibilities from the use of agar agar or similar substances in the treatment of habitual constipation. No claim is made that it will produce permanent cures, but surely no objection from any professional standpoint can be urged against the use of something, harmless in itself, which brings about a condition that approximates natural functions.

THE LOCATION OF THE CARDIAC APEX BEAT.

By Hughes Dayton, M.D.,

INSTRUCTOR IN PHYSICAL DIAGNOSIS IN THE CORNELL UNIVERSITY MEDICAL SCHOOL; ASSOCIATE ATTENDING PHYSICIAN TO THE NEW YORK HOSPITAL.

The easy recognition of certain auscultatory signs naturally distracts from other methods of physical examination of the heart. The beginner is known by the zeal with which he employs the stethoscope and dwells upon murmurs and rales; unfortunately, many never outgrow this stage, but use auscultation as a short cut to diagnosis. While recognizing the value of auscultation, we should not be led or misled by it and underestimate that of other diagnostic procedures. I desire to call attention to certain points in regard to the so-called apex beat of the heart.

The size and position of the heart are determined by: (1) The location of the apex as ascertained by inspection, palpation, or approximately, when these fail, by auscultation; (2) the area of relative cardiac dulness; (3) orthodiagraphy,[1] or radiography of the heart by a special apparatus, which employs only perpendicular rays, and so does not distort the cardiac outline. In describing the physical signs of the heart, no point is more loosely dealt with than the so-called apex beat. In some of the standard text-books one finds this term applied to the point of maximum visible pulsation in the

[1] Moritz, Münch. med. Woch., xlvii, 992.

lower portion of the precordium; in others, to that of maximum intensity of palpable impulse; while a few emphasize the most distant point of pulsation. In the records made by hospital internes one frequently finds noted P. M. I. (point of maximum intensity) so near the median line in a case of cardiac hypertrophy, especially of the right ventricle, as to show that the point mentioned is no indication of the situation of the apex. That many hospital internes seem so devoted to the term "P.M.I." shows that they have either been taught to regard the point of maximum impulse as the important point to localize or have so interpreted their instruction. Having noticed the emphasis laid upon the point of maximum intensity by medical students, hospital internes, and practitioners in New York City, I have investigated this point in all the text-books and manuals of medical and physical diagnosis and diseases of the circulatory system published since 1895, and some of earlier date, and found in the library of the New York Academy of Medicine. The contrast between the views of foreign and of the majority of these American writers is very marked.

In the text-book of A. L. Loomis,[2] we read: "When from any cause the impulse cannot be seen, its position must be determined by palpation." From this one would infer that seeing an impulse would obviate the necessity for palpation. As the visible impulse of the normal heart is not infrequently produced by a portion of the cardiac wall some distance internal to the apex, the impulse so spoken of is evidently the "point of maximum intensity," and this author, therefore, seems not to lay stress upon the observation of the apex beat as important in determining the size of the heart. That Hare[3] applies the term apex beat to the point of maximum visible pulsation is evident, for, after speaking of the apex beat as located two inches to the left of the sternum, he says: "Displacement to the right is due to adhesions and to hypertrophy and dilatation of the right ventricle, so that the apex beat is felt in the epigastrium or against the edge of the sternum." That, in employing the term apex beat, he refers to the point of maximum intensity is obvious, since in a case of hypertrophy and dilatation of the right ventricle the cardiac apex is really situated farther to the left than in a normal chest. So also Anders[4] says: "The precise apical pulsation may be localized by noting its point of maximum throb." However, he adds: "The extreme left border of the apical impulse in the fifth interspace extends nearly an inch beyond the actual maximum throb of the apex." He thus, emphasizes the manifestation of the site of the portion of the ventricular wall most prominent anteriorly rather than the extent of the ventricle toward the left, which is the important fact to be ascertained, since the situation of the apex beat is sought for in

[2] Lessons in Physical Diagnosis, 1899.
[3] Practical Diagnosis, 1907.
[4] Physical Diagnosis, 1907.

order to determine the extent of the heart toward the left and downward. His second statement, however, seems to show that he recognizes, although he fails to lay the chief emphasis upon, this important point. Da Costa,[5] Le Fevre,[6] and many others omit the term point of maximum intensity. Musser[7] says that a circumscribed area that rhythmically rises and falls is known as the apex beat, although it is a portion of the heart inside the true apex that strikes the chest wall. He makes no mention of the point of maximum intensity, and speaks of other areas of pulsation over the precordia as "new impulses." Butler[8] shows by his definition of the apex beat that he does not lay stress upon the maximum visible pulsation as Hare appears to do. After stating that the apex beat is somewhat internal to the apex formed by the ventricles, and that if there is much hypertrophy the enlarged and rounded heart strikes the chest wall, he adds: "The apex beat should be considered to lie at the point where the finger appreciates a distinct thrust from within." Another work which looks deeper than the visible pulsation is that of Gibson and Russell.[9] These writers say: "The normal apex beat is produced by the impulse of the apex of the left ventricle, but the term apex beat is very loosely applied, often used to denote the part of the precordia where pulsation happens to be most marked. It would seem better if, instead of stating that the apex beat is in such and such a space, it were stated that pulsation is visible in such and such spaces."

The best presentation of the subject in American works is that of R. C. Cabot,[10] who states clearly: "The maximum cardiac impulse is not due to the striking of the apex of the heart against the chest wall, but to the impact of a portion of the right ventricle. The importance of this fact is this: When we are trying to localize the apex of the heart, in order to determine how far the organ extends to the left and downward, it will not do to be guided by the position of the *maximum* impulse, for the apex of the heart is almost always to be found three-fourths of an inch or more farther to the left. This may be proved by percussion. The true position of the cardiac apex thus determined corresponds usually not with the *maximum* impulse, but with the point farthest out and farthest down at which *any rise and fall* synchronous with the heart beat *can be felt.*" It would seem more accurate to modify this statement to read: The point farthest downward and to the left at which a *distinct forward thrust of the heart* can be felt. This excludes the rise and fall of the chest wall, as a whole, which can often be seen and felt beyond the palpable thrust of the apex in cases with a heaving impulse which raises the chest wall. By careful palpation with a proper degree of pressure the limit, to the

[5] Principles and Practice of Physical Diagnosis, 1908.
[6] Physical Diagnosis, 1905.
[7] Medical Diagnosis, 1904.
[8] Diagnostics of Internal Medicine, 1901.
[9] Physical Diagnosis, 1893.
[10] Physical Diagnosis, 1905

left, of the clinical cardiac apex can often be distinguished as definitely as the edge of an enlarged spleen, provided it lies in an interspace and not behind a rib. We must acknowledge that the anatomical apex, formed by the tip of the left ventricle, being situated in a plane posterior to that of the right ventricle, comes into direct contact with the thoracic wall only when its apex is situated far enough to the left to strike the lateral wall of the chest.

Dietlen[11] says that the apex beat is almost always felt one interspace higher than the apex of the silhouette obtained by orthodiagraphy, or if the apex beat is under a rib, at least the width of one rib higher. He explains this by stating that the apex beat is felt in systole, while the silhouette shows the cardiac outline during diastole, and that while the heart changes only slightly in outline during the period of contraction and relaxation, such changes are greatest at the apex. It is the clinical apex, that portion of the heart wall situated normally about three-quarters of an inch internal to the anatomical apex, and the active cause of the palpable apex beat, that is of prime importance, since we must largely rely upon it for determining the size of the heart, orthodiagraphy being unavailable for routine examinations.

In marked contrast with the loose descriptions of some American text-books, apparently copied one from another, are the definitions almost invariably given in the works of German and British writers. Brugsch and Schittenhelm,[12] Ebstein,[13] Geigel,[14] Guttman and F. Klemperer,[15] and G. Klemperer[16] all define the apex beat as the portion of the palpable cardiac impulse situated farthest to the left and downward. As exceptions, we note Sahli,[17] who gives the Delphic definition of the apex beat as the visible and palpable lifting of the chest wall in the region of the apex; Edlefsen,[18] who appears to lay no special stress upon the extent of the heart to the left and downward; and Romberg,[19] who says that in cases with diffuse apex beat the apex is to be considered as situated at the point of strongest pulsation, or, if there is no point of maximum intensity, at the point farthest to the left.

Among the English and Scotch writers, Brown and Ritchie,[20] Sansom,[21] Sawyer,[22] and James MacKenzie[23] state definitely that the

[11] Deutsch. Arch. f. klin. Med., Band lxxxviii, S. 88.
[12] Lehrbuch der klinischer Untersuchungsmethoden, 1908.
[13] Leitfaden der ärztlichen Untersuchung., 1908.
[14] Lehrbuch der klinischen Untersuchungsmethoden, 1895.
[15] Ibid., 1904.
[16] Grundriss der klinischen Diagnostik, 1896.
[17] Lehrbuch der klinischen Untersuchungsmethoden, 1908.
[18] Lehrbuch der Diagnostik der inneren Krankheiten, 1899.
[19] Lehrbuch der Krankheiten des Herzens und der Blutgefässe, 1906.
[20] Medical Diagnosis, 1906.
[21] Diagnosis of Diseases of the Heart and Thoracic Aorta, 1892.
[22] Physical Signs of Diseases of the Thorax and Abdomen, 1908.
[23] Diseases of the Heart, 1908.

apex beat is the lowest and most external point of the cardiac impulse. Poynton[24] emphasizes the idea in these words: "In all cases a note should be made of *the extreme limit to the left* at which the impulse is palpable, and the maximum of the beat not alone relied upon."

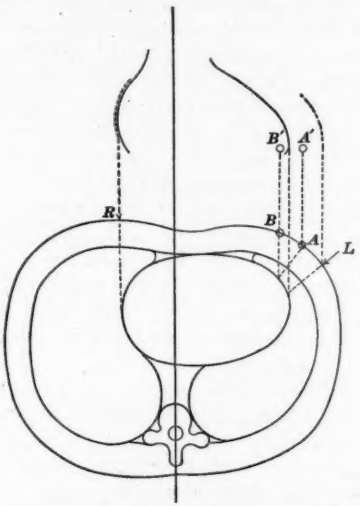
Barié[25] lays little stress upon the determination of the situation of the apex beat by palpation, on the ground that inspection is usually sufficient. He considers palpation as indispensable for finding the point of maximum intensity in cases of diffuse apex impulse.

Among the most important recent studies of the heart are those of Moritz,[26] and Dietlen,[27] who particularly oppose the determination of the size of the heart by the position of the apex beat and the left border of relative cardiac dulness, on the ground of their orthodiagraphic observations. They find that these physical signs, as determined by themselves, tend often to indicate a larger heart than really exists. Thus, Moritz[28] says: "By strong percussion one may very easily pass considerably beyond the left border of the heart," and, "the same is true if one uses as the limit of the left border of the heart the outer extremity of the apex beat, especially with vigorously pulsating hearts." (See figure.) Light percussion of the apical region and interpretation of the term apex beat, not as the point of maximum impulse or the most external portion of pulsation of the chest wall, but as the point farthest downward and to the left at which a distinct forward thrust of the heart can be felt, might obviate many of these discrepancies.

The importance of localizing the apex beat by palpation is that it is one of the two easily available methods of determining the extent of the heart to the left, the other, percussion, usually being of great value, but failing in cases with pericardial effusion, and in those in which the adjacent pulmonary resonance is obscured or lost through the presence of pleural thickening or effusion or pulmonary consolidation, through the presence of large breasts, or by the situation of the apex so far to the left, from any cause, that percussion elicits dulness of the lateral surface of the heart to the left of the apex. In such instances definite localization of the apex beat by palpation is of particular value, in the absence of deformities of the thorax, in determining the size of the heart or its displacement, the right border being located by percussion of the relative cardiac dulness. When an apex beat is visible it is usually even more definitely palpable, since firm pressure in an interspace may enable the observer to detect the impact of an apex situated so far from the chest wall as to strike it very lightly, especially during recumbency. In this connection it is important to emphasize the change in the relation of the heart to the chest wall, which occurs in moving from the recum-

[24] Heart Disease and Thoracic Aneurysm, 1907.
[25] Traité Pratique des Maladies du Cœur et de l'Aorte, 1900.
[26] Deutsch. Arch. f. klin. Med., lxxxi, 1; lxxxii, 1.
[27] Ibid., lxxxviii, 55.
[28] Münch. med. Woch., xlix, 1.

bent to the upright position. The cardiac apex is not only lowered,[29] but the apex, which is carried backward by gravity during recumbency, assumes a position nearer to the chest wall. For this reason palpation of the apex beat for the purpose of ascertaining the position of the apex is often easier when the patient is standing or sitting; occasionally the apex approaches the chest wall sufficiently to be palpable only when the patient bends forward. Percussion of the area of relative dulness must, of course, be performed while in the



Transverse section of the thorax (diagrammatic) seen from below, with the projection of the true outline of an enlarged heart (Moritz, Münch. med. Woch., xlix). Heavy percussion at *R* and *L* gives the area of dulness indicated by dotted lines in the projection and exaggerates the apparent size of the heart. The impulse felt at *A* and projected at *A'* is confused with the forward thrust of the apex felt at *B* and projected at *B'*, the clinical apex beat.

same position, in order that the outline of dulness, which also varies with change in position, may be considered in connection with the situation of the apex.

SUMMARY. The object of locating the clinical apex beat of the heart is to determine the size and position of that organ. The point of maximum impulse frequently does not define the site of the cardiac apex. The point of maximum impulse, either visible or palpable, may be produced by a portion of the heart wall at some distance from the apex. Except in cases in which the visible and palpable impulses coincide, as they usually do with a normal heart,

[29] Moritz, Dietlen, loc cit.

it is better to state simply that pulsation is visible in certain interspaces at a given distance from the median line. It is frequently impossible to determine the left border of the heart by percussion, on account of changes in the resonance of the adjoining area due to large breasts or intrathoracic conditions, or when the apex is carried so far toward the left by hypertrophy or by displacement of the heart, as a whole, that percussion, unless extremely light, tends to bring out dulness of the lateral surface of the heart. Auscultation, as a means of locating the apex, is at best a makeshift in the event of the failure of other methods.

The most valuable clinical method of locating the cardiac apex is the palpation of its impulse when this can be detected in an interspace. The apex beat should be considered as *the point farthest downward and to the left at which a distinct forward thrust of the heart can be felt.* The clinical apex so located, being close to the anatomical apex, is of real value in estimating the size or relative position of the heart. Because of the influence of posture upon the area of relative cardiac dulness, and the situation of the apex beat, it is advisable, for comparison with subsequent observations, to record whether the patient was examined in the erect or in the dorsal recumbent position.

ORTHODIAGRAPHY IN THE STUDY OF THE HEART AND GREAT VESSELS.

By Thomas A. Claytor, M.D.,
PHYSICIAN TO THE GARFIELD MEMORIAL HOSPITAL AND THE TUBERCULOSIS HOSPITAL, WASHINGTON, D. C.,
AND
Walter H. Merrill, B.L., M.D.,
RADIOGRAPHER TO THE GARFIELD MEMORIAL HOSPITAL, WASHINGTON D. C.

The *x*-rays, as ordinarily used, have proved of distinct value in the study of the intrathoracic vascular system, especially in the detection of aneurysms, but only to a small degree have they aided investigations of the heart itself. The difficulty before the perfection of the orthodiagraph has been the fact that, because of the divergence of the rays, the shadow of the object observed was enlarged. This exaggeration of outline is increased in direct proportion to the nearness of the object to the tube and its distance from the screen upon which the shadow is thrown. Thus, in order to estimate the size of the object under observation, an elaborate system of measurements is necessary, and even then accuracy is extremely difficult.

While it is true that the distortion of the image may be greatly diminished by taking the picture at long range, as it were, yet we lose the very important activity of life. Thus, with the fluoroscope the pulsations of the heart and aorta and the movements of the diaphragm serve as points of identification. Mediastinal tumors, calcareous glands, and tuberculous deposits in the lungs are not infrequently seen in close proximity to the heart and great vessels, and but for the pulsating movements of the latter could not be distinguished from them. It is the difference between the study of the living and the dead.

Description of the Orthodiagraph. The Röntgen-ray pictures are shadows, and, as the source of the rays is a point on the anticathode of the Crookes tube, these shadows would be the bases of cones or pyramids, whose apex is this point on the anticathode. The object casting these shadows would completely fill these cones or pyramids at some point between the apex and the base. Thus, as ordinarily used, skiagrams or Röntgen-ray shadows of the internal organs are always enlarged and distorted to a greater or less extent. To overcome this distortion and to outline the internal organs in their exact size, parallel instead of divergent rays should be used, and these parallel rays should be perpendicular to the reading screen. It is impossible to obtain parallel Röntgen rays directly from our Crookes tube, so that in the effort to overcome this difficulty the orthodiagraph has been developed.

The orthodiagraph, first devised by Moritz,[1] and later much improved by Levy-Dorn and Groedel,[2] is an instrument for outlining, in its exact size without distortion, any object that can be seen by the Röntgen rays. The working principle may be outlined as follows: A pneumatic pen, the centre of the anticathode of the Crookes tube, or source of the Röntgen rays, and the centre of a small fluorescent screen are so mounted on arms that they are in a straight line. These arms are attached to a carriage, which, while allowing all motions in one fixed plane, will keep this straight line perpendicular to the plane. Thus, the pencil of rays coming from the tube, through a small circular aperture in a lead diaphragm, to the centre of the screen, while it may be moved over a considerable surface, will always occupy parallel positions.

In using the machine it is only necessary to move the carriage so that the lead spot in the centre of the screen touches the edge of the shadow to be outlined; a mark is made with the pneumatic pen on the sheet of paper, which is clamped upon a drawing-board fixed behind the tube. The lead spot is then moved to a new position along this edge and a second mark made. In this way the process is continued until the entire shadows are outlined. Then, upon removing the sheet from the machine, these marks, when connected

[1] Münch. med. Woch., 1900. No. 29. [2] Orthoröntgenographie, München, 1908

will give the exact size of the object casting the shadow. The fluorescent screen may be removed from the arm to which it is attached and a pointer substituted. The pointer is so adjusted that it takes the place of the lead centre of the screen, and is, therefore, in the straight line with the centre of the pencil of rays and the pneumatic marker. It is used to outline the body and any points that the operator may wish to record upon the diagram sheet before the current is turned on.

Danger of *X*-ray Burns. The danger of burning the subject is practically nil. The tube is covered by a ray-proof shield and only the small pencil of rays in use is allowed to pass through a circular opening about 2 cm. in diameter in a lead diaphragm. As the apparatus is kept in almost constant motion, no area is exposed more than a few seconds at a time. The pencil of rays is thrown upon a fluorescent screen which is covered with a plate of lead glass, which it is claimed protects the operator's face and eyes while observing the shadow.

Advantages of the Orthograph. The satisfactory use of this instrument, as is the case with many others, such as the microscope or stethoscope, necessitates practice and a thorough familiarity with the normal. The orthodiagram, when completed, however, is intelligible to any one who takes the trouble to inform himself upon the topographical anatomy of the interior of the thorax. The lines are clearly marked and the object is outlined in its exact position with regard to its surrounding landmarks—the ribs, the sternum, the liver, the diaphragm—and its size may be determined and compared with the normal.

On the other hand, the skiagram usually requires long experience to interpret correctly. A shadowy distorted outline is often all that can be made out. This may mean much to the expert, but practically nothing to the ordinary physician, who must accept the statement of another and is often but half convinced. The fluoroscope, as ordinarily used, allows of no photographic or diagrammatic record.

Another great advantage possessed is the rapidity with which the diagram can be made when the operator has acquired some experience. Within a few minutes the complete examination is made, and the diagram is ready for inspection. The time and expense required for the development of a plate are avoided. The only delay is that necessary to allow the eyes of the operator to become accustomed to the darkness. This time varies from a few minutes to fifteen or twenty, depending somewhat upon the brightness of the day. We urge most strongly the absolute necessity of this delay, as without it the examination will be unsatisfactory.

Method of Making a Tracing. The patient may be placed in either a standing, sitting, or reclining position. The importance of keeping absolutely still must be impressed upon him, as any move-

ment of the body during the procedure will necessarily vitiate the results. The landmarks are then taken by means of the pointer before the current is turned on. They can be taken with the fluoroscopic screen, but the tube may as well be spared this unnecessary work. The midsternal line is fixed by marking the episternal notch and the tip of the xyphoid; the neck, shoulders, axillæ, and sides of the chest are outlined and the nipples (in males) marked. As many of the ribs as are desired may also be indicated. The dorsoventral illumination is the most satisfactory, and the sitting or standing posture is preferable when the subject is able to assume either of these positions. The heart's silhouette when thus observed consists of a series of curves, two upon the right and four upon the left side (Fig. 1). Upon the right, rising from the dark shadow of the liver, may be seen the "right auricular curve," (1) which is formed entirely by the right auricle. Above this is the "curve of the great vessels," (2) formed

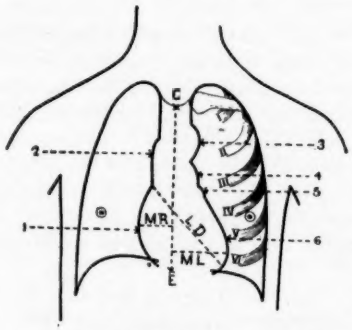


FIG. 1.—Orthodiagram of the normal heart.

by the descending cava or the aorta (in our experience this shadow is more often seen as a straight line). On the left, from above downward, is the "aortic curve," (3) formed by the arch of the aorta and its descending limb; below this is the "pulmonary curve," (4) formed by the pulmonary artery as it passes beneath the aortic arch. The next curve, the "left auricular," (5) is in our experience only exceptionally observed. It is produced in normal cases by the left auricular appendix, as only in abnormal conditions of the heart does the left auricle itself appear. The lowermost curve is the "left ventricular," (6) and is formed by the left ventricle.

It is convenient to trace the borders of the thoracic cavity (lung outlines), while the eyes of the operator are becoming accustomed to the darkness. Then the diaphragm is marked in its lowermost position during quiet respiration. The curves of the heart's silhouette are then outlined in the order indicated in Fig. 1. The right

border of the heart presents a well-marked dark shadow; the descending cava is lighter; the arch of the aorta, unless atheromatous, is often very faint, and could not be distinguished but for its pulsations; the pulmonary artery is faint, and the auricular appendix is not often distinguishable; the left border of the heart is clear and dark and in very active motion; the extreme apex is more difficult to see, because it is thin and also because its excursion of movement is large and rapid. By asking the subject to take a deep breath and hold it, the lower border of the apex can generally be outlined above the depressed position of the diaphragm. It must be borne in mind, however, that a deep inspiration alters the cardiac outline, that is, lowers the apex. The lower border of the heart is lost in the shadow of the liver, but its position can be easily estimated from the apical curve and that of the right auricle.

Observations. In order that we might properly appreciate diseased conditions, we undertook the study of a series of normal chests with the orthodiagraph. More than one hundred cases were studied, but from lack of sufficient data some of them have been omitted from our records. Our series of adults consists of 37 men, ranging in age from eighteen to fifty-five years, and 54 women ranging from nineteen to thirty-six years. Our measurements were made according to the accepted rules in this work. The method can best be understood from the diagram (Fig. 1). The total transverse diameter (T.D., the sum of M.R. and M.L.) may be considered as quite accurate, because the points from which these measurements are made are always clear and definite, but the longitudinal diameter (L.D.) is less accurate in some cases, because it is not always easy to tell where the right auricle ends and the descending cava begins. The extreme apex being thinner and in active motion renders the shadow at this point also less easy to trace.

Conditions Affecting the Heart's Size. It has been concluded from an examination of a large series of cases that the size of the normal heart depends upon a variety of factors. Thus, it depends upon the body height, the body weight, the age, the muscular development, the occupation, etc. Men, who have not attained full growth, even though of the same height and weight as adults, have smaller hearts. Further, the heart's size tends to increase in old age. Women of the same height and weight as men have proportionately smaller hearts. Finally, normal individuals presenting exactly like conditions may have different-sized hearts.

From our observations it appears that the size of the heart depends more upon the body weight than upon the body height. It was impossible in our series to work out a scale which seems to show any constant ratio between the heart's size and the body height. The body weight, on the other hand, compared very uniformly with the

heart's area. We have also constructed a similar chart from the measurements of Dietlen's[3] cases, which seems to agree with our findings.

TABLE I.—*Vertical Heart Orthodiagrams.* Male (37 Cases).

Weight pounds	Cases	M.R.	M.L.	T.D.	L.D.	
120–129	3	3.2	7.0	10.7	11.8	Min.
		3.7	7.2	10.9	12.6	Av.
		4.3	7.5	11.3	13.5	Max
130–139	5	3.5	7.5	11.0	12.0	Min.
		3.8	8.0	11.8	13.2	Av.
		4.2	8.5	12.5	14.0	Max
140–149	9	3.4	7.0	11.0	12.0	Min.
		4.0	7.7	11.9	13.4	Av.
		4.6	8.4	13.1	14.5	Max.
150–159	8	3.2	7.8	11.5	12.5	Min.
		3.9	8.4	12.3	13.5	Av.
		4.5	9.0	13.0	15.0	Max.
160–179	6	3.7	8.0	12.0	14.0	Min.
		4.0	8.2	12.4	14.6	Av.
		4.8	9.0	13.8	15.8	Max.
180–200	6	3.8	7.0	11.0	14.0	Min.
		4.2	8.7	12.9	14.7	Av.
		4.5	9.7	13.4	15.3	Max.

TABLE II.—*Vertical Heart Orthodiagrams.* Female (54 Cases).

Weight pounds	Cases	M.R.	M.L.	T.D.	L.D.	
100–109	2	3.2	6.7	9.9	12.0	Min.
		3.3	6.8	10.2	12.1	Av.
		3.5	7.0	10.5	12.3	Max.
110–119	3	3.0	7.0	10.0	11.5	Min.
		3.1	7.6	10.7	11.9	Av.
		3.2	8.0	11.1	12.4	Max.
120–129	14	2.3	6.4	10.2	10.5	Min.
		3.5	7.5	11.0	12.2	Av.
		4.2	8.6	12.2	13.8	Max.
130–139	19	3.0	6.4	9.6	11.2	Min.
		3.4	7.8	11.2	12.4	Av.
		4.0	8.8	12.6	13.3	Max.
140–149	5	2.6	7.0	10.0	12.2	Min.
		3.5	7.6	11.1	12.7	Av.
		4.1	8.3	11.8	13.2	Max.
150–159	7	3.1	7.6	10.9	12.3	Min.
		3.6	8.0	11.6	12.9	Av.
		4.8	9.3	12.8	14.2	Max.
160–175	4	3.5	6.5	10.6	11.8	Min.
		3.8	7.9	11.7	12.6	Av.
		3.8	8.5	12.3	13.0	Mean.
		4.1	9.0	12.8	13.2	Max.

[3] Deut. Arch. f. klin. Med., 1906–1907.

TABLE III.—*Vertical Heart Orthodiagrams.* Normal Cases. Males (42 Cases).

Age	Height in.	Weight Pounds	M.R.	M.L.	T.D.	L.D.	
18	62	125	3.0	7.0	10.7	11.8	Min.
32	68	150	4.0	8.1	12.1	13.7	Av.
55	74	200	4.8	11.0	14.0	15.8	Max.

TABLE IV.—*Vertical Heart Orthodiagrams.* Females (54 Cases).

Age	Height in.	Weight Pounds	M.R.	M.L.	T.D.	L.D.	
19	60	100	2.3	6.4	9.6	10.5	Min.
25	65	135	3.4	7.8	11.1	12.5	Av.
36	69	175	4.8	9.3	12.8	14.2	Max.

TABLE V.—*Dietlen's Horizontal Orthodiagrams.* Normal Cases.

	M.R.	M.L.	T.D.	L.D.
Average of 156 males	4.3	8.9	13.2	14.2
Average of 58 females	3.6	8.5	12.1	13.2

TABLE VI.—*Groedel's Vertical Orthodiagrams.* Normal Cases.

	M.R.	M.L.	T.D.	L.D.
Average of 60 males	4.6	8.4	13.0	14.0
Average of 54 females	3.9	8.0	11.9	12.9

TABLE VII.

	M.R.	M.L.	T.D.	L.D.	Area 70% of T.D. x L.D.
	M. F.	M. F.	M. F.	M. F.	M. F.
Dietlen's . .	4.3 (3.6)	8.9 (8.5)	13.2 (12.1)	14.2 (13.2)	131 (111)
Groedel's . .	4.6 (3.9)	8.4 (8.0)	13.0 (11.9)	14.0 (12.9)	127 (107)
Authors' . .	4.0 (3.4)	8.1 (7.8)	12.1 (11.1)	13.7 (12.5)	116 (97)

EXPLANATION OF TABLES AND CHARTS. Table I shows the heart measurements in our series of 37 men, whose average age was thirty-two years, average weight 150 pounds, and average height 68 inches, graded by weight. Table II shows the same arrangement of our series of 54 women, whose average age was twenty-five years, average weight 135 pounds, and average height 65 inches.

Fig. 2 shows graphically the increase of the heart measurements with the increase of the body weight in our series of 37 men. The upper line indicates the longitudinal diameter, the lower line the transverse diameter. These lines ascend by the scale of units given on the left of the chart, which expresses centimeters. The middle line indicates the product of L.D. by T.D., or approximately the square area of the heart's silhouette. (By a careful computation of the square area of the heart's silhouette in a number of diagrams from normal hearts, it was found to be about 70 per cent. of the product of L.D. by T.D.) This line ascends by the scale of units upon the right of the chart, which represents square centimeters. The reduction of the unit of measurement was necessary to keep this line within convenient limits.

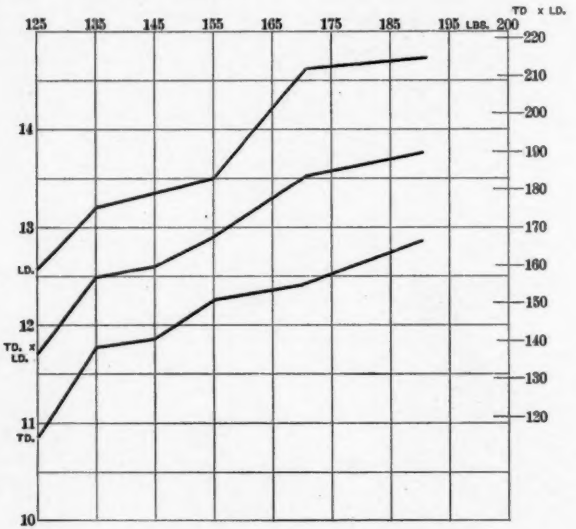


FIG. 2

It has been calculated from this chart that as the body weight of these men increased 60 per cent., the heart's area, as represented by the product of T.D. by L.D., increased 39 per cent. It is of interest to note that the size of the heart increased very rapidly up to 135 pounds of body weight, and that from that point it was more gradual.

Fig. 3 is the graphic arrangement of our 54 women. From this it would appear that as the body weight of these women increased 60 per cent., the heart's area, as represented by the product of T.D. by L.D., increased only 25.8 per cent., or far less rapidly than was the case with the men.

Fig. 4 is a graphic reproduction of Dietlen's table of horizontal

orthodiagrams of adult men grouped according to weight. The number of cases was 156, their average height was 169 cm.

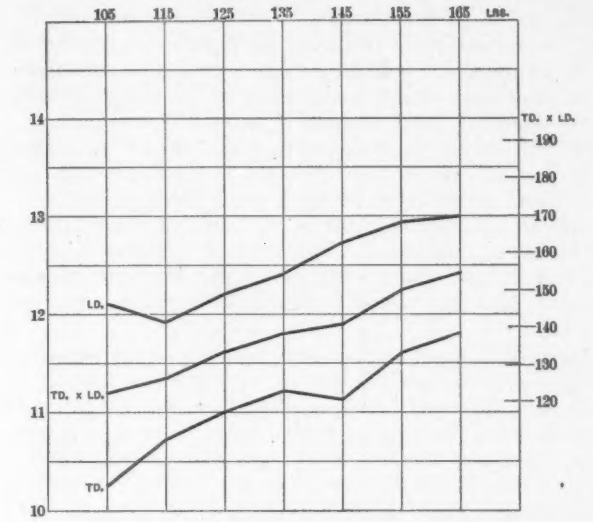


FIG. 3

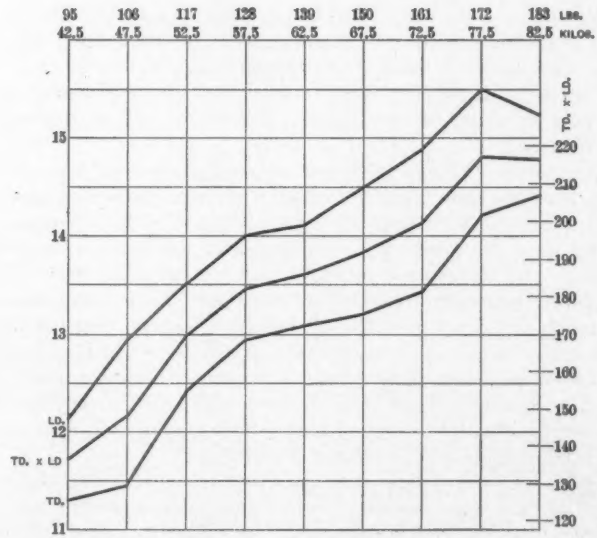


FIG. 4

(66.5 inches), and average age twenty-six years. This chart shows that as the body weight increased 60 per cent., the heart's area, as represented by the product of T.D. by L.D., increased 41 per cent., or practically at the same rate as was the case in our series of men. It is of interest to note in this chart the rapid rise in the heart's size from 95 pounds to 128 pounds, and the more gradual rise thereafter. The irregular appearance of the curve at the higher weights was probably due to the small number of men in these sections.

Table III shows the maximum, minimum, and average ages, heights, and weights of our series of 37 men, given in Table I, with 5 others added later, bringing the number up to 42. Table IV gives the same arrangement of our series of women. Tables V and VI explain themselves. Table VII is compiled from Tables IV, V, and VI, and shows in compact form the results obtained by Dietlen, Groedel, and ourselves in measuring the hearts of normal adults.

It may be seen that Dietlen's and Groedel's measurements are, as a rule, greater than ours, but this may be explained, perhaps, by the class of cases observed. Our series of men was made up almost entirely of brain workers in good health, and, as it is generally acknowledged that the amount of muscular exercise has a direct bearing upon the size of the heart, these men are likely to show slightly smaller measurements. It must also be borne in mind that Dietlen's measurements were taken from horizontal diagrams, which are somewhat larger than the vertical.

Our women were, for the most part, young and healthy, leading active lives requiring more or less muscular exertion. Their measurements come nearer to those of Dietlen and Groedel, but are also somewhat smaller. It may be seen, however, that the three sets of figures vary only by tenths of a centimeter, differences, almost too small to be considered.

Percussion. One of the chief advantages derived from the use of the orthodiagraph is the aid which it gives to those who make use of percussion in heart examinations. Many clinicians depend very greatly upon the percussion boundaries of the heart in making a diagnosis, and still more in giving a prognosis in certain heart lesions. It is pretty generally conceded by those who have compared their percussion findings with those of the orthodiagraph that the former are in many instances quite accurate, and that, as a rule, percussion may be depended upon for clinical purposes. At the same time it should be borne in mind that the men who have made these tests have been especially interested in this line of work, and have, therefore, become especially proficient in percussion.

Various methods of percussion have been used by various observers, with about the same results. The method made use of in our work has been "the immediate palpatory," as it may be called. Without going into detail, we would say that the percussion boundary of the

left border of the heart was usually found to be correct, but the right border was far more difficult to locate accurately. This has not been the experience of some other observers, as they have found the right border easier to determine. In almost all instances in which we felt satisfied with our efforts at percussion, the boundaries were found to

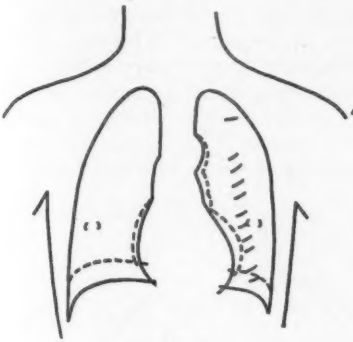


FIG. 5

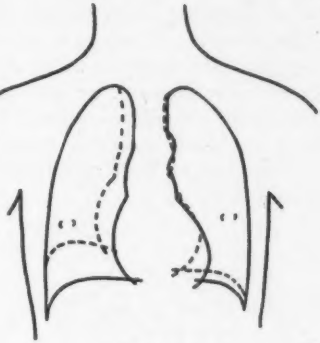


FIG. 6

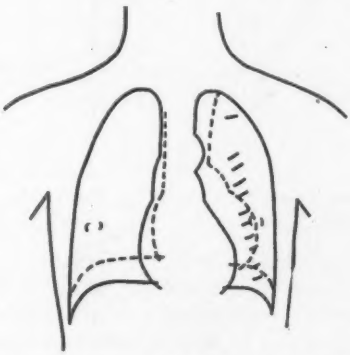


FIG. 7

FIG. 5.—The continuous line shows the position of the heart with the subject in the vertical position; the broken line shows it in the dorsal position.

FIG. 6.—The continuous line shows the position of the heart with the subject in the vertical position; the broken line shows it in the right lateral position.

FIG. 7.—The continuous line shows the position of t e heart with the subject in the vertical position; the broken line shows it in the left lateral position.

be correct, but in some subjects it was absolutely impossible to outline the heart by percussion, and this too in cases in which there was no excess of adipose tissue, or other demonstrable condition to interfere.

Figs. 5, 6, and 7 are orthodiagrams of a normal heart taken with the subject in the vertical, the dorsal, and the right and left lateral

horizontal postures. In each instance the continuous lines mark the position of the heart with the subject in the vertical position, while the broken lines show the effect of change of bodily position upon that of the heart.

THE FORM OF THE HEART'S SILHOUETTE IN DIFFERENT HEART AFFECTIONS. It has long been known that the hypertrophy or dilatation resulting from various heart lesions produces more or less characteristic alterations in the shape of the organ. This has been demonstrated upon the living subject many times by percussion, but in the orthodiagraph we have a far more accurate means of determining the change in outline. Our studies along pathological lines have not been extensive enough to enable us to speak authoritatively, but we hope in a future publication to go into this feature more fully.

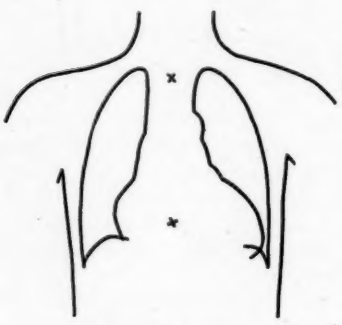


FIG. 8.—Orthodiagram of aortic insufficiency.

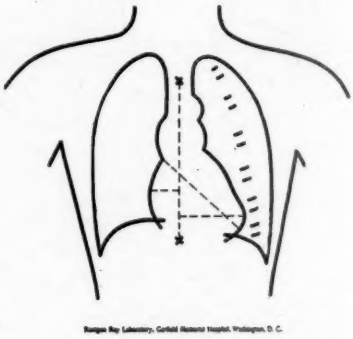


FIG. 9.—Orthodiagram of mitral stenosis.

For example, it may be seen from Fig. 8 that in aortic insufficiency the position of the heart is quite oblique, or rather foot-shaped; both diameters are greatly increased; the curve of the great vessels is more prominent, probably because of increased blood presure in the ascending limb of the arch of the aorta, or because of a dilatation of this section. The right auricular curve is prominent; the pulmonary curve is bulging; the left auricular curve is but faintly visible.

Of pure aortic stenosis we have no example, but the changes in contour are said to resemble very closely those of aortic insufficiency, only they are of less degree. The apex is more rounded, due to its increased breadth from the primary hypertrophy of the left ventricle.

Fig. 9 is from a case of pure mitral stenosis. The figure of the heart is more upright than in that of aortic disease; the curve of the great vessels is more pronounced than in the normal, and this we have observed in every case of mitral stenosis thus far examined by us. There is a decided bulging of the left auricular curve, due to the

dilatation and hypertrophy of this chamber of the heart. The left ventricular curve is relatively small, as might be expected.

Fig. 10 is from a case of pure mitral insufficiency. It is more upright than the aortic heart, but does not present quite the globular appearance which is said to be characteristic of this condition. The curve

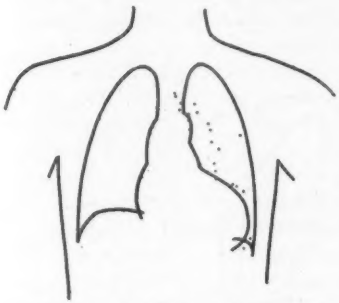


FIG. 10.—Orthodiagram of mitral insufficiency.

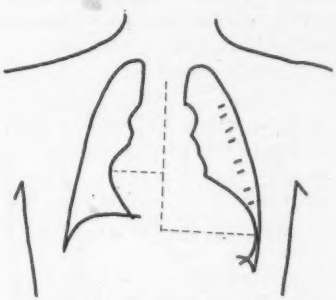


FIG. 11.—Orthodiagram of mitral and aortic insufficiency.

of the great vessels is prominent; the pulmonary curve is bulging, and the left ventricular curve has a tendency to rise higher toward the left axilla than in the normal.

Fig. 11 is a good example of combined mitral and aortic insufficiency. There is an enormous hypertrophy. The right auricular

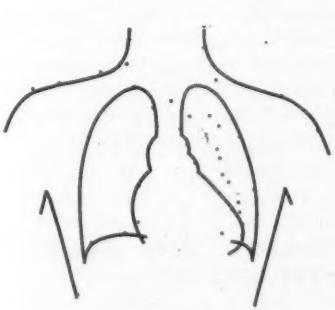


FIG. 12.—Orthodiagram of mitral insufficiency and stenosis.

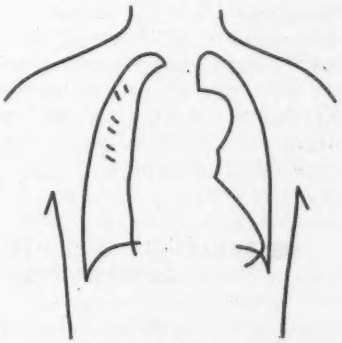


FIG. 13.—Orthodiagram of thoracic aneurysm.

curve is prominent, the curve of the great vessels is increased, while upon the left may be seen the step-like succession of bulging curves made by the aorta, the pulmonary artery, the left auricle, and, left ventricle. In other words, we have a combination of the distortions of outline produced in the conditions individually.

Fig. 12 is from a case of double mitral disease. The right auricular curve is marked, as is that of the great vessels; the pulmonary curve is bulging, and there is a decided prominence over the region of the left auricle, although it cannot be distinguished from the bulging curve of the left ventricle.

Fig. 13 shows a large aneurysm of the aorta.

Fig. 14 is an example of what has been called the "dropped" heart.

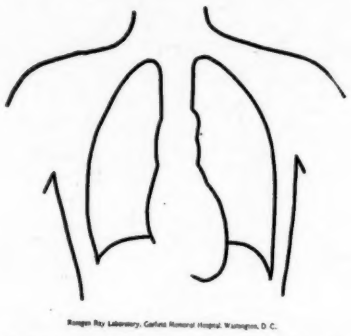


FIG. 14.—Orthodiagram of "dropped" heart.

CONCLUSIONS. While orthodiagraphy should not be looked upon in any way as a substitute for the other well-known methods of examination of the heart and great vessels, it is at the same time a valuable aid. It can be used to make fairly accurate outlines and measurements of the heart and great vessels, thus enabling us to make comparisons with the normal or with subsequent diagrams of the same case. The use of the orthodiagraph may also serve to prove whether or not the size of the heart is influenced to any appreciable degree by a single effort of exertion or by a single therapeutic or gymnastic treatment.

OBSERVATIONS ON ACUTE LEUKEMIA, WITH SPECIAL REFERENCE TO AUER'S BODIES.

BY REUBEN OTTENBERG, M.D.,
GEORGE BLUMENTHAL, JR., FELLOW IN PATHOLOGY, MOUNT SINAI HOSPITAL, NEW YORK.

OF the many supposed parasites that have been described in leukemia, none has been confirmed.[1] The bacterial infections which frequently occur in leukemia, and which are regarded by some as

[1] I have searched unsuccessfully for Löwit's bodies (Löwit, Central. f. Bakt., Orig., 1907, xlv, 600) in the blood, and for the spirochetes recently described by White and Proescher (New York Medical Journal, 1908, lxxxvii, 9) in the organs of one case of acute leukemia.

etiological, are undoubtedly intercurrent infections. Auer,[2] in 1903, observed certain peculiar structures in the large lymphocytes in a case of acute leukemia. The granules, crescents, and rods which he described were seen both in the fresh blood and in smears stained with various azure dyes or with Ehrlich's triacid mixture. These observations have been confirmed once by Pappenheim.[3]

Auer described three kinds of structures in the cytoplasm of the large lymphocytes—granules, crescents, and rods. Of these, the first two are indistinguishable from the azurophile granulations, now recognized as occurring regularly in lymphocytes.[4] The rods, on the other hand, have a distinct and characteristic appearance (Figs. 1, 2, and 3). They are found only in the cell body of the large lymphocytes. Generally they are single, sometimes two are seen (Fig. 4), and I have seen as many as four in one lymphocyte. In preparations stained by the Giemsa, Wright, or Hastings method they are red and remind one of the appearance of tubercle bacilli. Generally they are a little longer and more slender than the latter. Sometimes they seem nodular, generally the ends are tapering, sometimes abrupt.

Of the three cases reported here, I have seen only one myself. The other two were studied from smears in the blood collection of Dr. Libman. One of these two cases proved at postmortem to be a case of chloroma. The one case which I had an opportunity to follow was a typical case of acute lymphatic leukemia of the rapidly fatal type, with suppuration of the jaw, general glandular enlargement, large spleen, high continued fever, and multiple hemorrhages from the gums, intestines, and in the skin. The leukocytes varied between 170,000 and 225,000 in number, and the percentage of large lymphocytes was between 75 and 85.[5] Two blood cultures were negative. Cultures from the jaw during life and from the spleen postmortem showed Staphylococcus aureus. At autopsy the spleen was much enlarged (weight, 2 pounds, 7 ounces) and had numerous infarcts. There were many lymphoid infiltrations in the lungs. All the lymph nodes were enlarged and were greenish-gray in color. The bone marrow (femur) was light grayish brown in color and mushy in consistency.

The blood was examined for the rods from day to day. They were never present in anything like the large numbers in which they occurred in Auer's case (often four or five in a single microscopic field). It was generally necessary to examine one hundred to six hundred cells in order to a find a rod. No especial connection could be made out, such as there was in Auer's case, between the number

[2] Amer. Jour. Med. Sci., 1906, cxxxi, 1002. [3] Folia Hæmat., 1908, v, 347.

[4] Naegeli's statement that azure granules which are present in about one-third of normal lymphocytes are entirely absent in acute leukemia, is certainly incorrect.

[5] One interesting feature of the blood picture was the number of large lymphocytes whose nuclei looked as if dividing. One undoubted mitosis in the circulating blood was seen (Fig. 5).

FIG. 1

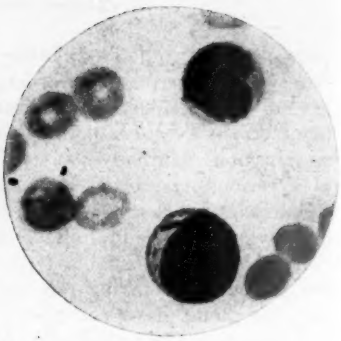


FIG. 2

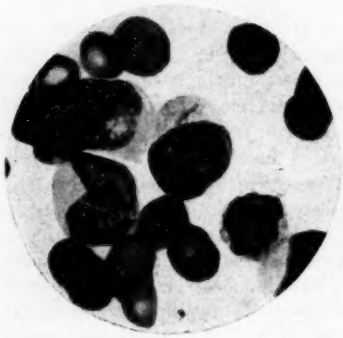


FIG. 3

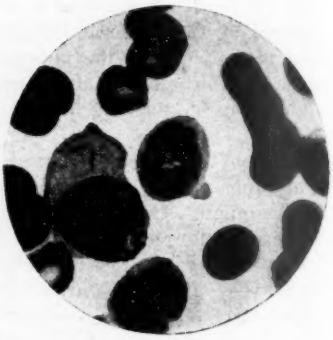


FIG. 4

FIG. 5

FIG. 1.—Auer body in a large lymphocyte. (× 1500) The granular appearance of the rod is well marked in the original print, but not well shown in the reproduction.

FIG. 2.—A rod somewhat shorter and thicker than those usually found. (× 1000)

FIG. 3.—A slender rod tapering at both ends, distinctly nodular in character. (× 1000)

FIG. 4.—Two rods in the same lymphocyte. (× 1000)

FIG. 5.—Mitosis in a large lymphocyte. (× 1000)

of rods and the hemorrhages. On three days no rods at all were found (as also happened in Auer's case).

In the other two cases, likewise, the rods were present in small numbers only. Of these two cases, one was remarkably similar in clinical characteristics to the case described above. It was also an acute case of three weeks' duration, commencing with stomatitis and suppuration of the lower jaw, continuing with high fever and general glandular enlargement. The number of leukocytes varied between 116,000 and 130,000, the large lymphocytes between 75 and 78 per cent.

The other case proved postmortem to be a case of chloroleukemia. It presented so many interesting features as to be worth describing in a little more detail. The total duration of the illness was three months. The symptoms were those of tonsillitis, with bone pains, progressive anemia, glandular enlargements, pleural effusion, and irregular fever. The white blood cells ranged between 25,000 and 30,000, the lymphocytes composing 90 to 97 per cent. of them. The large lymphocytes were 80 to 83 per cent. At autopsy the spleen was enlarged, there were enlarged mesenteric lymph nodes (one the size of an apple), of a grass-green color; the kidneys were enlarged and showed very numerous grass-green projecting tumors, varying in size and shape from a pin-point to 1 cm. in diameter. The marrow of the tibia, the only bone examined, was pale pink. On section of the green tumors in the kidneys and in the lymph nodes, they were seen to contain not only the usual large ungranulated mononuclear cells, but also large numbers of mononuclear cells with eosinophile granulations.[6] The contrast between the blood, which contained very few eosinophilic cells, and the tumors, which were full of them, was very striking. It seems probable that the green color of these tumors (which has never been adequately explained) may be due to the eosinophile granules, which in fresh preparations have a pale greenish tinge.

In studies of this kind the control observations are of great importance. Do these rods occur in the lymphocytes in any other disease, or in health? I have examined the blood of many normal subjects, both infants and adults, as well as of many other types of disease, including myelogenous and chronic lymphatic leukemia. No rods were found. It should be remembered, however, that in the three cases of leukemia in which the rods were found it was necessary generally to search through several hundred large lymphocytes in order to find a rod. In acute lymphatic leukemia this is easy. But in normal blood or the blood of other diseases the large lymphocytes form so small a proportion of all the cells that it takes hours to see as many cells of this variety as can be seen in a few minutes in a case

[6] The occurrence of eosinophile cells in chloroma has been described by Dock and Warthin, Medical News, November, 1904, and by F. F. Leighton, Jour. Path. and Bact., October, 1907.

of acute leukemia. I have, however, examined many hundreds of large lymphocytes from a rather remarkable case of chronic lymphatic leukemia which is at present under observation. In this case the number of large lymphocytes is high, at times as high as 86 per cent., and an examination of these cells made by Dr. Weil according to the new oxidase method[7] indicates that they are of myelogenous origin. In this case the rods have not been found.

I have also recently examined the blood of a case of acute leukemia of the aplastic type in a child. Though the clinical symptoms in this case were those of a typical and very acute lymphatic leukemia, during the days which the child spent in the hospital before its death the white blood cells were never above 12,000, and the large lymphocytes 16 per cent. (though the small lymphocytes were as high as 86 per cent.) Streptococci were isolated from the blood. No rods were found.[8]

The interpretation of these rods is difficult. They might be regarded as: (*a*) Artefacts; (*b*) cell secretions; (*c*) parasites. That they are artefacts can be excluded. They have been seen in the fresh blood (Auer and Pappenheim). They occur only in one type of cell and only in one place in that cell; and they can be stained by a number of different methods. They have a definite and regular size and shape. Whatever they are, they are real. The question of these rods being products of cell activity cannot be answered definitely at present. They may possibly belong to the same order of things as the azurophile granules seen in many lymphocytes. They may be crystals. (They, of course, have nothing to do with Charcot crystals, which[9] occasionally occur not only in the tissues, but in the blood of leukemia.) The fact of their occasionally being found in vacuoles of the cell body might be regarded as being in favor of their excretory nature. They are not likely to be products of cell disintegration, as they are usually found in well-preserved cells. There is no proof that they are parasites. Attempts made to cultivate them, from the blood of the patient, on agar, bouillon, ascitic fluid, and sodium citrate, were all negative. As the leukocytes in these nutritive media degenerated and lost their staining power the rods also disappeared.

It was hoped that some evidence on this question might be obtained from the organs postmortem, but examination of smears from the spleen, bone marrow, lymph nodes, and other organs failed to show

[7] Schultz, Ziegler's Beiträge, 1909.

[8] This case probably belonged to the very interesting group of "aplastic leukemias," of which cases are described by Emerson. (Johns Hopkins Hosp. Bull., 1907, p. 82), Hand (Arch. Ped., December, 1905), Wolff (Berl. klin. Woch., 1905, xlii, 35), and Jeanselme and Weil (Bull. et mém de la soc. méd. des hôp. de Paris, 1904, xxi, 185). It may be, however, that cases of this kind are really acute bacterial infections, and that the blood picture is secondary. "It is certain that true septicemias can produce a blood picture quite like acute leukemia" (Emerson, loc. cit., p. 73).

[9] Wells, Chemical Pathology, p. 259.

the rods or anything like them. This negative finding, however, should not count for too much, as the cells in the smears postmortem were all degenerated and stained poorly, whereas in the blood rods were seen only in perfectly preserved lymphocytes. (Auer did not obtain an autopsy. Pappenheim does not state in his description whether the bodies were found in the organs postmortem or not.)

An effort was made in one other way to throw some light on the question—namely, by inoculation. There have been many unsuccessful attempts at inoculation of leukemia, not only from man into the lower animals,[10] but from man to man (Schupfer)[11] and from dog to dog (Weil and Clerk[12]). Recently Ellerman and Bang[13] have reported the successful inoculation of the true leukemia of the fowl and its transmission through several generations of fowls.

In the present experiments macacus monkeys were chosen. One monkey received intravenously 15 c.c. of blood drawn directly from the patient. The other received in the same way 10 c c. of spleen pulp from the same patient postmortem. Both monkeys have been kept under observation for six months and have apparently remained healthy. The blood of both monkeys has been examined frequently and has at times shown a marked relative lymphocytosis, small lymphocytes predominating. The experiments are to be regarded as negative, and it seems probable that leukemia, like malignant tumors, cannot be inoculated from one species of animal into another.

CONCLUSIONS. 1. The rods described by Auer can be found in many cases of acute leukemia.

2. They have never been found in any other disease.

3. There is no evidence that they are parasites.

4. The attempted inoculation of human leukemia into monkeys was unsuccessful.

5. Leukemia (like tumors) can probably only be successfully inoculated, if at all, into animals of the same species.

I wish to thank Dr. F. S. Mandlebaum for his kindness in making the accompanying photographs.

[10] Teichmüller, Deut. Arch. f. klin. Med., 1907, p. 62.
[11] Review in Jour. Amer. Med. Assoc., 1905, p. 882.
[12] Arch. de méd. exper., 1904, xvi, 462.
[13] Centralbl. f. Bakt., 1908, xlvi, 595.

VOLKMANN'S CONTRACTURE.

By Nate Ginsburg, M.D.,

ASSISTANT DEMONSTRATOR OF ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA; CLINICAL ASSISTANT SURGEON TO THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE.

Richard Volkmann,[1] in 1869 and later in 1875, first described a deformity of the hand and wrist resulting from some interference with the blood supply of the flexor group of muscles of the forearm, usually following the application of splints for fracture occurring in the region of the elbow-joint. The involvement of nerve trunks was mentioned, but to these structures the principal changes were not attributed. The condition is usually styled Volkmann's ischemic paralysis, in order to attribute the profound anatomical changes producing this deformity to the existence of a primary myositis of ischemic origin, due to various factors surrounding the individual case. Writers earlier than Volkmann are said to have called attention to this condition, and to Leser[2] undoubtedly belongs the credit of having emphasized the importance of this type of deformity prior to Volkmann's more definite description.

From a review of the literature on the subject which has appeared since Volkmann's first publication, one is led to believe that this deformity is of rare occurrence. Dudgeon,[3] in 1902, searched the literature and reported only fifteen cases. He apparently failed to collect cases occurring outside of England. Powers[4], in 1907, collected 52 cases, and Taylor[5], in 1908, reported 59 cases, but failed to include two cases reported by Ward. Following the completion of this paper comes the last report by Thomas[6], of 107 cases, to which I can add the case operated upon by Taylor and the one to be reported in this paper. This brings the total number of cases of which we have authentic record up to 109. While it appears that this condition is one of rare occurrence, we are led to believe, however, that many more cases have been observed than have found their way into printed reports. Surgeons to many hospitals with large surgical clinics have had the misfortune to observe this deformity, resulting from either the injury itself or the treatment instituted to immobilize the parts. The printed reports of cases come principally from England, Germany, and this country; so that it is apparent that many cases may have occurred in other countries which have never been reported. Following Volkmann's original publication, attention was directed to this subject by the English

[1] Billroth's Path. u. Chirurgie, 1869, ii, 846; Centralbl. f. Chirurgie, 1881, viii, 801.
[2] Volk. Sammlung. klin. Vorträge, 1884, No. 249.
[3] Lancet, 1902, p. 78.
[4] Jour. Amer. Med. Assoc., 1907, xlviii, 759.
[5] Annals of Surgery, September, 1908, 394.
[6] Ibid., March, 1909, 330.

physicians, notably Littlewood,[7] Page,[8] Barnard,[9] and Dudgeon, the latter having written quite at length on the deformity from a neurological standpoint.

There is by no means a unanimity of opinion as to the causative factors producing this deformity, which is not always a true paralysis. Some observers have believed various accidents attendant upon the injury to be the cause of the resulting contracture, while others believe that the malformation is the result of improper treatment, that is, too much splint pressure.

If we term this deformity and physical disuse of the hand, which, as a rule, is out of all proportion to the degree of injury sustained, Volkmann's contracture, we are not bound to confine the primary anatomical changes solely to the muscle tissue. Whether the condition is the result of the profound ischemia of the flexor and pronator muscles of the forearm, resulting in a myositic degeneration with subsequent scar tissue formation due to a mechanical obstruction of the arterial blood supply to this group of muscles, or whether it be due to a disturbance in the integrity of the vascular structures owing to injury sustained by the trauma producing the fracture (Bloodgood), the type of deformity is usually the same. That this condition of marked contracture and loss of flexor motion of the fingers in the affected limb is not always due to a primary myositis, to the exclusion of other causes, I shall show from the case here reported, in which muscle changes were of secondary importance compared with the disturbance in the continuity of the nerve structures.

Definition and Cause. Volkmann's contracture is essentially a deformity of the fingers of the hand, in which the wrist-joint takes part, and comes on rapidly with loss of muscular power of the forearm. Two cases out of 109 have been observed in which the contracture affected the foot, due to injury and involvement of the flexor muscles of the leg. The condition is almost invariably associated with a fracture of the forearm or humerus in the region of the elbow-joint. It is usually observed in young children under the age of fifteen years, although Dudgeon treated a case occurring in a man of twenty; and Wallis[10] and Ward[11] each observed a similar deformity in women of this same age. Slöffer[12] reported a remarkable case in a boy, aged eighteen years.

The fundamental factors producing the condition are interference with the circulation of the muscles of the part, and injury to the nerve structures in close relationship to the involved muscles. That the condition of contracture as seen in the well-developed type is not always primarily of myositic origin is shown by cases following the prolonged application of an Esmarch bandage above the elbow-joint, and by Slöffer's case, in which thrombosis of the axil-

[7] Lancet, 1900, 290. [8] Ibid., 1900, 83. [9] Ibid., 1901, 1138.
[10] Practitioner, 1901, lxvii, 429. [11] Lancet, 1902, 372.
[12] Wien. klin. Woch., January 3, 1901, 24.

lary artery followed a gunshot wound of the thorax. The ball lodged in the axilla, and injured the artery.

Dudgeon reported pressure sores in seven out of fifteen cases, due to splints too tightly applied. Taylor states that at least 80 per cent. of reported cases have resulted from splint pressure for fractures of the forearm or humerus. Davies-Colley[13] observed pressure sores occurring in his cases, and ascribed this as the specific cause of the contracture. Page believed the deformity to be the result of a combination of pressure, fixation, and ischemia. Although Volkman stated that he had never seen a case result from the use of an Esmarch bandage, but knew it to be a cause, the case reported by Wallis, in which an Esmarch bandage was applied for over an hour, is proof of the influence of this factor in the production of this condition. Anderson believed the prolonged fixation of fractures of the forearm by any form of apparatus that interrupted the free circulation of blood through the muscles and nerves of the part acted as the causative influence producing this type of contracture. Bloodgood[14] doubts the myositis theory resulting from anemia of the muscles, believing that, although such cases undoubtedly do occur, the probabilities are that more cases are due to injury of the bloodvessels at the time of the accident.

It is curious that with the tight application of splints, usually an anterior and a posterior, to the extent of producing a pressure sore, the muscles involved should always be the flexor group, to the total exclusion of the extensors. It is due to this fact that the typical contracture is produced. That the extensors are not, as a rule, involved may be explained on the following anatomical grounds. On the anterior surface of the forearm the soft structures overlie the radius and ulna, forming at the internal aspect of the upper forearm quite a pad of soft tissue. In addition to this arrangement, the body of the flexor group of muscles is chiefly anterior, coming in direct contact with the splint when applied. The relation of a splint on the dorsal surface of the forearm is such that the ulna is interposed between it and the soft tissues, chiefly the extensor group of muscles. It is to the interposition of this bony structure, which undoubtedly receives the splint pressure, that the extensor muscles owe their escape when splints are so tightly applied as to cause a tissue necrosis with the production of a sore. While this reasoning seems adequate to explain the deformity as a direct result of faulty splint adjustment, it does not definitely solve the factors producing a contracture after long application of an Esmarch bandage, or a direct injury to the vessels some distance from the site of the muscle changes. It is reasonable to expect as much damage to the extensors from these causes as the flexor group suf-

[13] Guy's Hospital Gazette, 1898, n. s., xii, 460.
[14] Progressive Medicine, December, 1908.

fers, and yet such does not appear to be the case. It is therefore apparent that some other factor must obviously enter into the causative influences producing this deformity. Since no other structures are more exposed to injury than the nerve trunks with their close proximity to the arteries, a constriction sufficient to close the large vessels of the brachial region lasting over a long time must necessarily exert marked pressure on the median and ulnar nerves, and induce a neuritis with subsequent nerve degeneration. That the extensor muscles do not suffer is due to the fact that the musculospiral nerve, which ultimately distributes branches to this group of muscles, is deeply placed in the upper arm between the triceps muscle and the humerus. In the region of the elbow-joint the nerve is protected by its position between the long extensors of the forearm. It is, therefore, not subjected to as much trauma as the other cords because of their superficial position in the upper arm.

Embraced as it is by the pronator radii teres muscle, the median nerve is practically subjected to the same forces inducing injury as is this structure. It would seem, therefore, that if pressure over this muscle and its associated group is sufficiently great to cause a profound ischemia, the nerve must primarily share the fate of the muscle which encloses it, and suffer degenerative changes to some extent. Sensation of the fingers does prove that the nerve is involved in very many instances, and the occurrence of flexor muscle paralysis with no marked myositic changes strengthens this view. If we accept a primary myositic and neurogenic origin for this type of contracture, we are able to bridge over deficiencies in the causative factors occurring in cases in which the integrity of the muscle structures is less disturbed than that of the nerves. In this way can be explained those cases of Volkmann's contracture in which no fracture existed, or in which no profound changes took place in the muscles.

Symptoms. The symptoms are usually of rapid occurrence, "coming on in half a day or less in very severe cases" (Volkman); but they may be delayed over a period of many days. The promptness with which the hand assumes the condition of contracture, with the characteristic deformity of the fingers, is surprising. If splints have been applied there is usually considerable pain in the forearm and fingers, due to the great pressure, which is augmented by the traumatic swelling of the parts. Some difference of opinion seems to exist in regard to the presence of pain in these cases before the immobilization of the limb is relieved. Dudgeon believes pain is conspicuously absent in most cases, and attributes the progression of the deformity to the lack of interference due to no complaint on the part of the patient. Tightly applied splints for any injury usually cause discomfort, and it is reasonable to suppose that when splints are so firmly adjusted as to produce a pressure sore, the patient will feel decided discomfort and much pain until

their removal. This appears to be the experience of most observers of this condition.

It is important to remember that the condition varies in severity in direct proportion to the length of time the splints are allowed to remain applied, and the degree of pressure and injury sustained. It is due to this fact that much difference of opinion exists regarding the prognosis and treatment, because one case may be irreparably damaged before splint removal, while another with prompt treatment may be of a mild type with apparent functional recovery.

The characteristic deformity is usually that of a pronated forearm slightly flexed at the elbow. There is a decided flexion of the hand on the wrist-joint, and an extension of the metacarpal-phalangeal joints. The interphalangeal joints of the fingers and the terminal joint of the thumb are flexed, and the fingers are often buried in the palm of the hand. Extension of the fingers is impossible with the hand extended at the wrist by any justifiable force, and when the wrist is sharply flexed, the extension of the fingers can usually be procured, although in very severe types even complete extension cannot be made in this position. When pronounced myositis has occurred, later there is usually loss of the soft resiliency of the flexor group of muscles, and a greater degree of resistance is present with a board-like sensation. Add to this condition a cicatrized sore at this site, and the thickening of the tissues may be very marked.

Pressure sores may occur on the flexor surface of the upper forearm, and on either aspect of the wrist. In the case here reported, the photographs show the large shell-like scars on both surfaces of the wrist region. The anterior sore involved the superficial flexor tendons and the median nerve, causing a dissolution of their continuity. Schram reported 60 per cent. of pressure necrosis of the skin in his series. If a pressure sore is present, it usually occurs over the flexor surface of the forearm. Splint pressure may also rarely produce a sore on the posterior surface of the forearm near the elbow. These wounds may not always do so, but depending upon their severity, the necrosis may involve nerve and tendon structures and lead to a pitiful case. From his observation of cases of this severe type, it is possible that Davies-Colley ascribed the contracture to the result of a pressure sore.

Before the splints are removed, marked discoloration and swelling of the hand may be observed with bleb formation on the fingers. In old cases the loss of the use of the hand and nerve involvement will produce a shining, glossy skin, which is cold to the touch; if sensation is impaired, accidental burns may result, as occurred in the case presently to be reported. Trophic changes are absent in this condition, and ankylosis of the joints, although sometimes apparent, has never been observed in a true Volkmann's contracture. Shortening of the limb, which is not of infrequent occurrence in cases of long standing, is suggested by Turney as being due to

the lack of growth of the bones owing to the diminished support they are required to afford the affected muscles.

NERVE REACTIONS. The normal electrical reactions of both muscles and nerves is thought by Oppenheim[15] and Turney[16] to be an important diagnostic factor. Dudgeon and Hedley[17] believe with Turney concerning this feature of the disease. Depending upon the time of observation and the duration of the condition when examined, the results of electrical examinations of the muscles and nerves will greatly vary and therefore account for the different expressions of opinion. Involvement of nerve trunks was found to exist in sixty-two of the one hundred and seven cases contained in the table of cases collected by Thomas. He believes this lesion was probably present in some of the other cases, as in many papers the details of the examinations were not given.

PATHOLOGY. The histological changes causing this deformity are still a matter of some speculation. Volkmann believed that the deprivation of oxygen to the muscles owing to obstruction of the blood current caused a condition of muscle asphyxia with the subsequent production of a condition simulating rigor mortis. The venous stasis which occurs simultaneously with the occlusion of the arterial current hastens the onset and adds to the severity of the deformity. Raymond Johnson[18] and Riedinger[19] believed the splint-pressure inducing an anemia of the part produced an inflammation and subsequent fibrous degeneration of the muscles. Davies-Colley thought the condition could be largely accounted for by attributing the pathological changes to the pressure sore invading the soft parts of the flexor region of the upper arm. Wallis and Ziegler[20] account for the structural changes by extravasations of blood serum and round cells into the tissues affected, with subsequent fibrous tissue proliferation, causing contracture of the muscle substance. Kraske[21] thought extreme cold could produce this deformity; and Hildebrand[22] and Bloodgood believe that some direct injury or interference with the blood current at the time of the accident, rather than splint pressure, is the underlying cause in most of these cases. Little mention seems to be made of nerve injury, and yet an analysis of the cases with the symptoms resulting from the injury shows early and often severe nerve involvement. Hildebrand suggested that nerve changes might be primary in their origin, but his view was not shared by Bardenheuer,[23] who thought that nerve degeneration followed secondarily. No doubt many cases will show nerve destruction at the time of the accident, in addition to the profound histological changes in the muscles.

[15] Diseases of the Nervous System, 1904.
[16] Quoted by Dudgeon, Lancet, 1902, p. 78.
[17] Ibid., p. 82.
[18] Lancet, 1898, i, 722.
[19] Sitzungs. Berichte, der phys.-med. Gesellsch. zu Würzburg, 1902, No. 3, 33.
[20] Lehrbuch der Allg. Path., etc., p, 306.
[21] Centralb. f. Chir., 1879, No. 12.
[22] Deut. Ztschr. f. Chir., 1890, xxx, 98.
[23] Centralb. f. Chir., 1906, xxxiii, 505.

The nerves at operation are often found swollen and thickened or so degenerated as to present only a fibrous strand of tissue. Some cases at operation have shown a wasting of the muscles with a pale and dry appearance.

In the case reported herewith the muscle substance which was exposed at operation showed no macroscopic changes which would indicate injury or degeneration. In this case the deep pressure sore on the anterior surface of the wrist had invaded all the superficial flexor tendons and the median nerve. The nerve had been destroyed at the middle of the forearm, and the distal end could not be found at the wrist. Most of the superficial tendons had also been lost through infection.

Diagnosis. A fracture of one or both bones of the forearm or of the lower extremity of the humerus occurring in a child under fifteen years of age accompanied by much swelling, with the immediate application of splints, usually of the anterior and posterior variety, resulting in discoloration of the hand with the presence of much pain, and a tendency for the fingers to contract, should at once suggest the possibility of the presence of a Volkmann's contracture. Removal of the splints will at once present the characteristic features of this deformity, especially if added thereto a pressure wound is seen. Volkmann stated that the paralysis and contracture arise simultaneously, not as in paralysis of nervous origin, in which contraction is a later phenomenon and of gradual onset. While the contracture, as a rule, is of rapid occurrence, the hand assuming the position shown in the photographs, the deformity may be somewhat delayed by the continued use of an anterior splint after the fingers have begun to contract.

At times some difficulty may arise in differentiating a Volkmann's contracture from other conditions in which pure nerve lesions are the underlying causes. In ulnar and median nerve paralysis the prompt loss of sensation in structures to which these nerves are distributed will result. Muscle paralysis will follow at once, depending upon the group of muscles to which the nerve distributes branches. In ulnar nerve paralysis the reactions of degeneration in the wasted muscles are present, but there is no reaction to stimulation with the faradic current. The resulting atrophy of the muscles of the hand, and the development of the so-called "claw-hand" in paralysis of this nerve, will clearly indicate the nature of the lesion.

Trophic lesions may be present, and there is usually no flexion of the wrist in nerve palsies. In musculospiral nerve paralysis there is usually a bilateral wrist-drop, unless the nerve on one side is injured from the surface. The muscles involved in this type of deformity are the extensors of the forearm, and the lesion is easy of recognition.

Prognosis. "There is no more pitiful condition than this after an injury, usually a fracture, in healthy strong children"

(Bloodgood).[24] The prognosis in cases in which the hand and fingers are affected with much deformity is bad, and if nerve involvement is present, as appears to be the case in many instances, the outlook for a good functional result is practically hopeless. Anderson[25] opposed Volkmann's gloomy prognosis and believed that function of the hand and fingers could in a large measure be restored, but he did not cite any cases to substantiate his claim.

The extent of the damage done to the muscles and nerves will in a very large measure determine the outcome of the case, if prompt treatment is instituted. It is due to this fact that early cases with little time for extensive changes to occur have yielded to measures tending to restore the normal tone of the structures involved. Some cases are undoubtedly hopeless from the beginning, and no form of treatment will preserve the use of the affected hand.

TREATMENT. There is apparently wide difference of opinion in regard to the course of treatment to be pursued for the purpose of restoring the hand to its normal appearance. So great has been the difference of opinion as to the specific underlying cause of this type of contracture, and the proper treatment to be undertaken, that we are given Volkmann's almost hopeless prognosis on the one hand, and later the hope expressed by Anderson that it was the exceptional case which did not recover. This difference of view is obviously due to the varying degrees of injury which may be sustained, resulting either in a deformity of almost transient type or one of hopeless contracture and physical disability. When little splint pressure has existed, with only a slight or no break in the continuity of the skin, and prompt recognition of the condition (with measures for relief), the resulting deformity will not be of the type generally seen after the long application of splints with deep pressure wounds involving the muscles, tendons, and nerves. In the latter class of cases any form of treatment will often be of little use, and hence the choice of the many methods of correcting the deformity will vary with the individual case and the skill of the operator. A careful review of the cases tabulated by Dudgeon, Ward and Powers, and later by Thomas, will show some of the best results following an expectant line of treatment supplemented with massage, passive motion, and electricity.

It matters little which factor is foremost in producing this deformity, or whether it can only occur from splint-pressure, and not result from too long application of an Esmarch bandage—the fact remains that the disuse and appearance of the hand resulting from either cause conforms with the contracture first noted by Volkmann, and the correction of the condition confronts the surgeon, who is left to select the operation which best suits the individual case.

[24] Progressive Medicine, December, 1908.
[25] The Deformities of the Fingers and Toes, p. 66.

In spite of the greatest care, splints properly applied for a fracture of the forearm or humerus near the elbow will often, in a few hours, become tight and uncomfortable from the swelling and œdema of the part. It is, therefore, a wise precaution to see all fracture cases within a few hours after the reduction of the fracture and the immobilization by splint application. If there is discomfort, prompt removal of the dressing and its reapplication, allowing for the swelling of the part, will be the best prophylactic means of obviating the development of this deformity.

The evidence adduced by the experimental work of Lapinsky[26] and Leser[27] tends to prove that the continued immobilization, following tight splint pressure with resulting ischemia of the muscles of the part, is a very important factor in the production of this deformity. Therefore, with the symptoms of this condition, the prompt removal of the splints or plaster bandage is of the first importance. Every effort should be made toward restoring the hand to its normal anatomical and physical appearance. The splints may be left off if necessary, allowing the fracture to care for itself. Passive motion, massage, electricity, and even active motion must be resorted to at intervals of a few hours to correct the condition. A deformity due to the improper reduction of the fracture is of much less significance compared with the severe result from the reapplication of splints if the hand and wrist are already contracted.

Once present, the condition must be carefully studied with a view to determining the extent of muscle, nerve, and tendon involvement. If large and deep pressure sores are present, little can be done until they are healed. When tendon and nerve structures are much involved, and subsequent destruction results, due to infection of the wound, the outlook is almost hopeless from any standpoint, and the loss of time awaiting the healing of the wound is not a great factor in the treatment.

If pressure sores are slight and heal quickly, treatment should be resorted to promptly.

Non-operative Treatment. There are some cases which will probably recover without operative treatment. This class includes cases with slight injury to the muscles and nerves with little resulting deformity, and in which there has been prompt recognition and treatment as suggested. The treatment is often tedious, discouraging at first, and must be carried out over a long period of time, often many months, and sometimes years. In this class of cases the principle measures to be employed are massage, passive and active motion, and electricity, with permanent removal of the

[26] Deut. Ztsch. f. Nervenh., 1900, xvii, 322.
[27] Samml. klin. Vorträge, Leipsic, 1884, p. 249.

splints if deemed necessary. Such cases have been reported by Leser, Niessen,[28] Dudgeon, and others.

The extension of the fingers and hand under an anesthetic as the sole measure of treatment, employed by Volkmann, has proved valueless, and often difficult to accomplish. Mechanical extension of the fingers and hand over a long period of time (Sayre[29]), and continuous elastic traction (Martin[30]), have been employed with good results. Robert Jones,[31] of Liverpool, obtained a good result by separate splinting of the fingers, with flexion at the wrist. By increasing the length of the splints he was enabled eventually to secure extension of the fingers. He believes this gives better results than operative treatment The various means of treatment with the success attained by each tends to prove the assertion that all grades of injury and deformity may occur, to which special types of treatment are adapted.

Operative Treatment. Garré,[32] in 1895, and later Henle[33] and Raymond Johnson, first advised resection of portions of the radius and ulna in order to restore the hand to its normal position. By shortening the forearm the flexion of the fingers and hand at the wrist are overcome and motion is permitted, provided the muscles have not undergone complete fibrous transformation.

In shortening the bones care must be taken to perform the resection at different levels to avoid fixation of the bones from the union of the callous in the interosseous space. This operation is short, and simple in its performance, and has given very good operative relief in many cases in which it has been tried. The danger of non-union (Raymond Johnson) is slight, but shortening of the arm is produced, and if there is much growth of the bones later, some contracture may recur (Erdman[34]).

Tendoplasty. This operation has been successfully employed to overcome the marked flexion present. The English surgeons, particularly Page, Littlewood, Barnard, Wallis, and Ward, have done this operation with marked success, in some cases almost completely restoring the use of the hand. This operation has many warm advocates, and is preferable to resection of the bones in some cases, particularly when the tendons and nerves are involved in the newly formed fibrous tissue affecting the muscles, or in the scar resulting from a pressure sore.

Lengthening of the tendons is a tedious and often difficult operation to perform, but allows of an exploration of the injured struc-

[28] Deut. med. Woch., 1890, xvi, 796.

[29] Mentioned by A. S. Taylor as presented before the Pediatric Section of the New York Academy of Medicine, March 12, 1901.

[30] Congress Français de Chirurgie, Paris, 1903, xvi, 934.

[31] Amer. Jour. Orthop. Surg., 1908, v, 377.

[32] Deut. med. Woch., 1895, xxxi, 484.

[33] Henle. Quoted by Dudgeon.

[34] New York Surgical Society, April 8, 1908, quoted by A. S. Taylor.

tures and a transposition of the ulnar and median nerve if either is found too tightly in the grasp of the scar tissue.

The danger of mistaking tendons and accidentally injuring the nerves in the forearm are somewhat exaggerated, and the chief difficulty in the performance of this operation is the length of time required. If the scar resulting from the pressure sore is large, it should be excised and transplantation of a skin flap done, the upper abdomen being a convenient region from which to utilize the necessary tissue. The latter method was pursued by Dr. Stewart in the case here reported.

Tenotomy. Tenotomy has been practised in a few instances to overcome the deformity present. This measure eliminates all future possibility of obtaining any use of the hand, and while it improves the appearance of the limb, it is doubtful whether this procedure should be followed even after every possible means has been tried to restore the function of the fingers.

These are the principle operative procedures that have been tried for relieving Volkmann's contracture. Prompt relief to nerves which appear to be involved in the scar tissue should be afforded and superficial transplantation done, as recommended by Kleinschmidt[35] and Freeman,[36] who believe in very early nerve involvement in these cases. Bardenheuer believes the nerve changes in these cases are secondary to muscle involvement, and advocates an operation for the purpose of separating the muscles.

Whatever operation may be done to cure the condition, every assistance should be given the patient, following recovery from operation, to restore the use of the hand. For this purpose massage, electricity, and active and passive motion should be tried, and carried out over a long period of time. It is surprising the improvement that will follow after a long course of the above treatment, covering many months and sometimes years. In addition to these means, congestive hyperemia preceding the bath and massage is recommended by Taylor.

The following case was observed and studied by me: J. S., a male, aged twelve years, a school-boy, was first seen on August 30, 1908, in the out-patient surgical clinic of the Philadelphia Polyclinic, in the service of Dr. Francis T. Stewart, to whom I am indebted for the privilege of studying and reporting the case. There are no incidents in the medical history of the case previous to the accident which have any bearing upon the present condition. On August 20 the boy fell out of a second story window and sustained a fracture of the right radius about 3 cm. above the wrist-joint (Colles' fracture). He was treated in the hospital surgical clinic about forty-eight hours after the injury was sustained, when the splints (both an anterior

[35] Centralb. f. Chirurg., xxxiv, 81. [36] Surg., Gynec., and Obs., July, 1907.

and posterior) which were applied within an hour after the fracture were removed because of great pain, swelling, and discoloration of the fingers of the hand. There were a number of blebs on the palmar surface of the thumb. He had suffered continual pain following the reduction of the fracture and the application of the splints, which increased in severity and then gradually disappeared, to be followed by numbness of the part. Upon removal of the splints, the right hand was observed to be swollen, discolored, and somewhat colder than its fellow. There was a little flexion of the hand on the wrist, and some contraction of the interphalangeal joints of the fingers. The thumb was adducted, and three or four blebs containing bloody serum were present. The deformity was not very marked, because the splints had been very tightly applied by means of strips of adhesive plaster and a bandage of muslin. Over the flexor surface of the wrist there was a large oval pressure sore extending into the subcutaneous structures. A similar sore was present on the dorsal aspect of the wrist, but did not involve the extensor tendons. A slight break in the continuity of the skin just below the elbow-joint, where the upper edge of the posterior splint had pressed, was present, with two or three blebs. There was no pressure wound over the body of the flexor muscles of the forearm.

Some flexion of the forearm at the elbow-joint was present and persisted for several weeks. Motion of the fingers and the hand was very slight, and painful. Skiagraphic examination revealed a fracture of the radius about 3 cm. above the wrist joint, with some anterior projection of the lower fragment.

The posterior splint was removed permanently, and every effort was made to heal the large pressure sore on the flexor surface of the wrist. Any motion of the fingers or hand was attended with such excruciating pain that for the first week the wounds on both surfaces of the wrist were dressed daily and lightly bandaged to an anterior splint extending to the tips of the fingers. The progression of the deformity was rapid, and by the end of the first week the characteristic pronation and flexion of the hand, with contracture of the fingers described, was present. The splint was removed and kept off, and since nothing could be done until the deep wounds healed, the limb was dressed daily and carried in a sling. Infection of the sore on the flexor surface of the wrist followed, and the superficial tendons sloughed away, leaving a thick contracted scar, as shown in the accompanying photographs (Figs. 1, 2, and 3). The healing of this wound occupied many weeks.

At the time the electrical examinations were made by Dr. Spiller, November 15, 1908, the deformity was very marked, and corresponded to the classical description of Volkmann's contracture. The following is Dr. Spiller's report: "J. S. has prompt reaction to faradic current in the right forearm, in the muscles of both flexor and extensor surfaces. There is slight contraction of the flexor

and extensor muscles below the wrist. Sensation for touch and pain are greatly impaired below the wrist and apparently normal above the wrist."

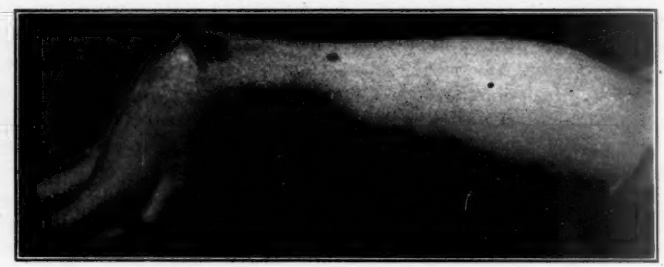


FIG. 1.—A large scar on the dorsal surface of the hand; flexion of the hand at the wrist, and of the fingers at the interphalangeal joints.

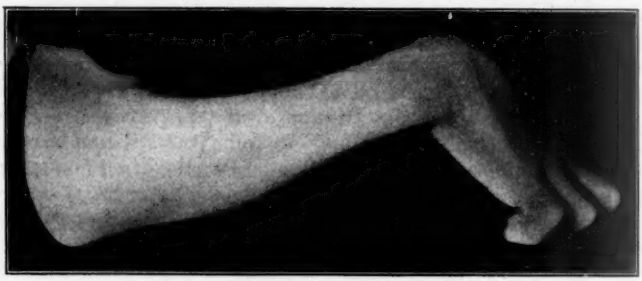


FIG. 2.—Marked pronation of the hand; flexion of the forearm on the elbow, and extension of the fingers at the metacarpal-phalangeal joints.

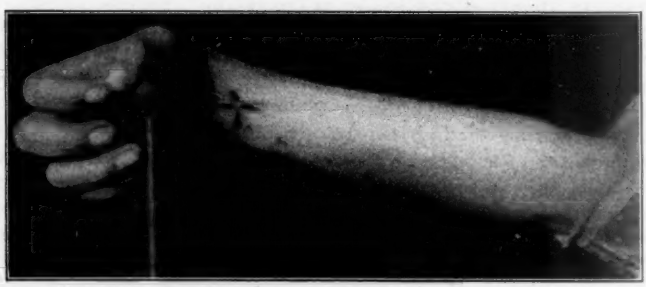


FIG. 3.—A large scar on the anterior surface of the wrist, resulting from the pressure sore; the fingers and thumb present the characteristic contracture observed in this type of deformity.

So greatly was sensation impaired in the fingers of the hand that an accidental burn occurred over the first interphalangeal joints without the knowledge of the patient.

When the pressure sores were finally healed, the hand presented practically a useless appendage, and the tips of the fingers were almost buried in the palm. With complete flexion of the hand on the wrist, the fingers could not be fully extended, and pronation of the hand was marked. Passive and active motion had only succeeded in overcoming the flexion at the elbow.

It was apparent that this case presented a combination of many factors, producing the deformity: involvement of nerve, tendon, and probably muscle structures.

Operation, November 23, 1908, by Dr. Francis T. Stewart. The operation was performed under ether, and required over two hours. Excision of the scar on the anterior surface of the wrist was performed, leaving a large denuded area. The longitudinal incision was carried up to the middle of the forearm, and a careful dissection of the adherent and fibrous structures was made. The tendons of the flexor sublimis digitorum and palmaris longus muscles were absent, and all that remained was their proximal ends. The ulnar nerve was intact and uninjured, and the pulsation of the ulnar artery was feeble. The pronator quadratus was so changed as to present merely a white quadrilateral fibrous structure with no macroscopic remains of muscle tissue. The median nerve was destroyed from the middle of the forearm to below the annular ligament at the wrist, and only the proximal end could be found where it emerged from the mass of the pronator radii teres muscle. The nerve seemed to be continuous with a thin fibrous strand of tissue. The radial pulse was also feeble.

The anterior projection of the lower fragment of the radius formed a sharp ledge, over the border of which the structures were pressed by the tightly applied splints. It is quite likely that this feature of the accident greatly assisted in the subsequent destruction of the tendons and median nerve which followed the primary injury. Incision carried to the flexor muscles showed no macroscopic alteration in their appearance suggestive of a very pronounced myositis.

Tendoplasty was performed, utilizing the deep flexor tendons and the remains of the superficial group of tendons. The fingers and the hand were extended, and the forearm placed midway between pronation and supination. Transplantation of a flap from the epigastric region, leaving one end attached, was done to fill in the large gap left by the removal of the scar on the flexor surface of the wrist. The hand was bandaged across the upper abdomen for ten days, at the end of which time the attached end of the flap was cut and the hand freed.

Postoperative Course. The graft, consisting of the skin and subcutaneous fatty layer, healed promptly and was not followed by

a contracted scar. At the end of two weeks active and passive motion were begun, and supplemented with massage, electricity, and baths, which are being continued at the present time. Contracture of the fingers has recurred, but the deformity is not as severe as it was previous to the operation. The thumb is still adducted, but allows of slight motion, and there is some improvement in the extensor and flexor movements of the fingers. Sensation in the hand has distinctly improved, but the limb is very far from being a useful extremity. At this writing, steady improvement has been noted in the recovery of the motion of the fingers. It is to be hoped as time goes on that enough motion of the fingers and hand may be secured to enable the patient to perform the more necessary acts.

In this case we have the contrast of a careful painstaking operation, the purpose of which was to free any adherent structures in the forearm, and to lengthen the tendons, thereby correcting the deformity of the hand, with the operation of resection of portions of the radius and ulna recently performed by Taylor. The operation done in this case was tedious and time consuming, but it seemed preferable to any other procedure. In both cases the ultimate result was poor, owing to the incurable state of the damaged structures. Although improvement is slowly going on, it is extremely doubtful whether in either case complete recovery will ever take place.

Conclusions. 1. The specific underlying factor producing a Volkmann's contracture is still a matter of dispute; but the evidence seems to warrant the assumption that it is a deformity in which muscle, nerve, and tendon structures share. Early, and probably primarily, the nerve structures share the same injury which induces muscle anemia with subsequent myositic degeneration. The contracture is the result of a myositis, with accompanying nerve lesion, and may vary from a transient type to that of hopeless deformity.

2. This deformity follows, in most cases, too tightly applied dressings for fractures of the forearm or humerus in the region of the elbow-joint, in children under the age of fifteen years. Usually the pressure has been of sufficient intensity to produce a pressure sore, which is present in 80 per cent. of the cases. It may arise within less than twenty-four hours, or be delayed for many days.

3. Severe pain, followed by numbness, discoloration of the hand with bleb formation, and contraction of the fingers are first noticed. Later, the characteristic flexion of the fingers at the interphalangeal joints, with flexion of the hand at the wrist, and marked pronation occur. Some flexion at the elbow is usually present.

4. Careful application of dressings, with frequent inspection, is the best prophylactic measure. Prompt recognition of the symptoms, with measures to restore the hand to its normal appearance,

constitute the safest means of obviating the subsequent development of what may prove to be an irremediable condition. Active and passive motion, massage, and electricity, with permanent removal of the splint, should be advised if deemed necessary.

5. Operative treatment depends upon the individual case. No specific procedure can be suggested, but tendoplasty and resection of both bones of the forearm are the two methods of operative treatment offering the best chance of recovery of use of the hand. The nerves should always be examined to determine the extent of involvement, and should be freed if necessary.

6. The prognosis is unfavorable depending upon the duration of the case and degree of involvement of the damaged structures. Partial recovery is not unusual in cases seemingly hopeless in the beginning.

A CASE OF APPENDICITIS IN WHICH OXYURIS VERMICULARIS WAS FOUND IN THE APPENDIX.[1]

By Astley Paston Cooper Ashhurst, M.D.,
SURGEON TO THE OUT-PATIENT DEPARTMENT OF THE EPISCOPAL HOSPITAL, PHILADELPHIA.

Laura P., a Polish girl, aged fourteen years, was admitted on March 14, 1909, to the Episcopal Hospital, in the service of Dr. G. G. Davis, to whom I am indebted for the privilege of operating and of recording the case. Owing to the patient's ignorance of the English language, it was difficult to obtain an accurate history, but it was learned that she had been ailing for a week or ten days, and that twenty-four hours before admission she had been taken with severe abdominal pain, followed shortly by vomiting. The pain was at first general, but later settled to the region of the appendix. Examination at 9.30 P.M., soon after admission, showed great tenderness with some rigidity over the right iliac region. No mass was present. The temperature was 100.6° F., the pulse 88, and the respirations 34 per minute. The white blood cells numbered 25,000 per c.mm. The diagnosis was acute appendicitis.

Operation at 10 *p.m.* Through the transverse incision of G. G. Davis,[2] an appendix was removed which was congested at the tip, but presented no other gross evidences of disease. The abdominal wound was closed without drainage. When the appendix was slit open at its tip, the lumen of the organ was found to contain a number of minute worms, resembling "seat worms." Subsequent examination under the microscope by Dr. C. Y. White, director of

[1] Read at a meeting of the College of Physicians of Philadelphia, April 7, 1909.
[2] Annals of Surgery, 1906, xliii, 106.

the laboratories of the hospital, confirmed the diagnosis of Oxyuris vermicularis. When warmed up, the worms squirmed around actively. Under the microscope the appendix presented the ordinary picture of acute suppurative appendicitis in the early stage, its walls being studded with miliary abscesses. The patient's convalescence was uneventful, and examination of her feces revealed the presence of many more similar parasites.

My object in reporting this case is to call attention to the fact that the presence of intestinal parasites in the appendix is by no means rare, but that they are seldom, if ever, the direct cause of appendicitis. As I have pointed out in the chapter on the History of Appendicitis, prepared for the third edition (1905) of Dr. John B. Deaver's monograph, *Fabricius ab Aquapendente*, even as early

Vermiform appendix containing Oxyuris vermicularis. (Natural size.)

as 1634, mentions having found at times a worm in the appendix at autopsy; Santorini (1724) made the same observation, and thought that the chief function of the appendix was to serve as a nest for round worms, where they might be cherished and be prevented from escaping into the general intestinal tract.

Many varieties of parasites have been found in the appendix, especially Ascaris lumbricoides and Oxyuris vermicularis. F. D. Patterson[3] has collected ten cases in which lumbricoid worms and eight in which oxyurides were found in connection with diseased appendices. Tapeworm, trichocephalus, echinococcus, and bilharzia disease were also noted, in one instance each. To his list of oxyuris should be added two cases, reported by Beyea[4] and by Rammstadt.[5]

Still,[6] in 200 consecutive autopsies on children less than twelve years of age, found oxyurides in the intestinal tract in thirty-eight cases (19 per cent.), and of these cases no less than twenty-five, or two-thirds, had the parasites inside the appendix. Oppe, according to Sprengel[7], found oxyurides six times among sixty appendices removed at operation. Erdman[8] found, among 29 operations for

[3] Amer. Jour. Med. Sci., 1906, cxxxi, 859.
[4] Univ. Med. Mag., 1900, xiii, 67.
[5] Deut. med. Woch., 1902, xxviii, 919.
[6] Brit. Med. Jour., 1899, i, 898.
[7] Appendicitis, Deutsche Chir., Stuttgart, 1906, S. 182.
[8] New York Med. Jour., 1904, i, 537.

appendicitis in children under ten years of age, 4 cases with oxyurides in the appendix. Rostewsew, cited by Sprengel, found parasites three times in the appendix among 278 autopsies on persons who had not died of appendicitis; and also found them present the same number of times among 163 cases of appendicitis: from which he concluded that their influence in causing appendicitis was trifling.

It is well known that L. Metchnikoff holds parasites responsible for the majority of attacks of appendicitis; and he insists that in every case, before operation is undertaken, a microscopic examination of the feces should be made, and that in cases in which it is possible the effect of vermifuge remedies should be tried, even if this examination is negative. In 1901,[9] when he appears first to have called attention to this matter, he referred to four cases in which recurrent attacks of appendicitis ceased after lumbricoid worms had been expelled by the action of vermifuges. He acknowledges that it is not at all probable that the lumbricoids could perforate the appendix or other portion of the intestinal canal, but explains their action on the theory that they first produce erosions of the mucosa, and then deposit in these erosions the microörganisms with which their bodies are covered. In this way, Metchnikoff contends, parasites can be the primary cause of ulceration and perforation of the intestinal wall.

Yet I think the case reported herewith goes to show that even if the worms can be held accountable for the origin of the disease (which is a mere assumption), the lesions in the appendix, even at an early stage, are such as to make prompt operation the safest form of treatment. In this case the walls of the appendix were found on microscopic examination to be studded with miliary abscesses, and it was the opinion of the pathologist that the presence of the worms was a mere coincidence; delay in operation in this, as in most other cases, would have resulted in a localized peritonitis within a comparatively short space of time. From the fact that intestinal parasites are found within the appendix in 12.5 per cent. of children under twelve years of age (Still), it is not logical to conclude that they are on that account frequent causes of appendicitis. It is interesting to note their frequency, and it is important to take measures for their extermination; but the surgeon should not waste valuable time in attempting to cure symptoms of appendicitis by vermifuges, when it is not proved that the worms, even if present, are responsible for the symptoms of disease.

[9] Journal des Praticiens, 1901, xv, 185.

REVIEWS.

MODERN MEDICINE. ITS THEORY AND PRACTICE. In Original Contributions by American and Foreign Authors. Edited by WILLIAM OSLER, M.D., Regius Professor of Medicine in Oxford University, England, assisted by THOMAS MCCRAE, M.D., Associate Professor of Medicine and Clinical Therapeutics in Johns Hopkins University, Baltimore. In seven volumes. Vol. VI, Diseases of the Urinary System, of the Ductless Glands, of the Muscles, Diseases of Obscure Causation, Vasomotor and Trophic Disorders, Medical Aspects of Life Insurance. Pp. 799. Philadelphia and New York: Lea and Febiger, 1909.

OF the many and important subjects discussed in Volume VI of Osler's *Modern Medicine* major interest attaches to diseases of the urinary system and of the ductless glands—which together comprise about one-half of the volume.

The book opens with an introductory chapter by John McCrae on the normal and diseased physiology of the kidney, which summarizes well what is known of the subject, and is followed by an instructive chapter on malformations and circulatory disturbances of the kidney. A. E. Garrod contributes a chapter on anomalies of urinary excretion, in which, among other matters, he makes brief reference to tests of the functional competency of the kidneys, and discusses well such conditions as albuminuria, hematuria, pyuria, etc. In another chapter he tells us what is really known of uremia, and if he has been unable definitely to state the immediate cause of uremia, he has mentioned with sufficient critical comment the existing theories, except, perhaps, that which predicates major significance to the liver, and he describes accurately the clinical manifestations and the treatment of the condition. The important subject of nephritis, or Bright's disease, is discussed in a chapter of over one hundred pages by James B. Herrick; and to this he adds a short discussion of amyloid kidney. His discussion of the, or a, classification of the nephritides is rather illuminating; his opinion regarding the chronic cases is, perhaps, well reflected in his statement that "much energy is wasted and much needless disappointment experienced in the attempt to make a given case fit into a classification that is largely artificial and necessarily imperfect." He finally distinguishes a chronic parenchymatous, a chronic interstitial, and a mixed type, and of the

chronic interstitial, a primary, a secondary, and an arteriosclerotic form. In discussing the treatment of acute nephritis he pertinently points out the irrationality of giving large amounts of water with the idea of flushing the kidneys, emphasizing the fact that since the damaged and inflamed kidneys need rest, they refuse to excrete the excess of water which, retained, usually serves only to augment the œdema. His own practice is to be guided by the thirst in giving water, and this, perhaps, is as good a practice as any. The undoubted clinical value of a sodium-chloride-poor diet in œdema is also pointed out, without commitment to the theory that chloride retention is the cause of the œdema or that which looks upon the chloride retention and the œdema as phenomena of nephritis not related as cause and effect. The old-fashioned Basham's mixture is the author's favorite preparation of iron—when iron is needed. The pathogenesis of the cardiovascular changes seems still to be in an unsatisfactory state. Thomas R. Brown contributes interesting chapters on the bacteriology of the infections of the urinary tract and the urinary findings in these infections, on pyogenic infections of the kidney, ureter, and perirenal tissues, and on tuberculosis of the kidney. The general excellence of the chapters precludes any adverse comment, although perhaps it would have been well to accentuate somewhat the unilateral hematogenous infections of the kidney that recently have been submitted to clinical study, and are sometimes recognized and cured by operation. Tumors of the kidney, urinary lithiasis, and diseases of the prostate are discussed by Hugh H. Young—than whom it is doubtful if any one is better qualified. His observations on genito-urinary diagnosis may be read with profit by all practitioners.

The section on diseases of the ductless glands is contributed by George Dock. The discussion of Addison's disease, Graves' disease, cretinism, myxœdema, tetany, akromegaly, etc., is all that the most critical reader may expect. Considerable attention is devoted to disordered function of these ductless glands and to aberrant or ill-developed diseased manifestations, so-called *formes frustes*—which are all too commonly overlooked. The discussion of the treatment of exophthalmic goitre will afford some satisfaction to the conservative clinician who hesitates to hand over to the surgeon all his cases without a thorough trial of efficient, medical, remedial measures; these are well discussed and offer considerable hope of a successful issue in many cases. The indications as well as the dangers of operative treatment are also pointed out. The section is altogether worthy of its author—who contributes also a short article on osteomalacia.

The editors themselves contribute to this volume: Dr. Osler, articles on Raynaud's disease, angioneurotic œdema, and diffuse scleroma, and Dr. McCrae, an article on arthritis deformans.

Dr. Osler's name has long been identified with at least two of the diseases that he describes; the present articles may be viewed as authoritative pronouncements on the subjects. Dr. McCrae has handled very well the intricate subject of diseases of the joints commonly described under the designation arthritis deformans, but also passing current under a diversity of other names. Our real ignorance of the subject, especially of the etiological factors, is answerable for the existing complexity of the discussions and unsatisfactory classifications, and if Dr. McCrea has not brought order out of chaos, he has told us about all that is known on the subject, and what he says is based upon a large personal experience.

There are other articles of value: Warfield T. Longcope describes Hodgkin's disease, with painstaking investigations of which his name has become identified; Daniel J. McCarthy discusses astasia-abasia, adiposis dolorosa, myasthenia gravis, paramyoclonus multiplex, and periodic paralysis—intricate subjects well described; Walter R. Steiner writes on myositis, Thomsen's disease, and myotonia congenita; and Charles P. Emerson describes achondroplasia, hypertrophic pulmonary osteoarthropathy, Paget's disease, osteogenesis imperfecta, osteopsathyrosis, leontiasis ossea, microcephalus, and facial hemiatrophy. The volume is concluded with a really excellent discussion of the medical aspects of life insurance by Charles Lyman Greene.

As a whole, the volume contains a relatively large number of articles of diversified interest; a high level of general excellence is maintained throughout in the discussions—which for a long time must remain authoritative. A. K.

Appendicitis and other Diseases of the Vermiform Appendix. By Howard A. Kelly, M.D. Pp. 489; 215 illustrations, 3 lithographic plates. Philadelphia: J. B. Lippincott Company, 1909.

Dr. Kelly explains succinctly the purpose he had in view in the preparation of this new edition of his monumental work on the vermiform appendix, by saying that after the publication of the former it soon became evident that a compact resume dwelling with especial care on the practical side of the subject would better meet the daily needs of the great army of general surgeons throughout the country. His preface to the first edition is republished, although some of it is quite inapplicable to this book. The statement that he has "to thank Dr. John B. Deaver, of Philadelphia, for the use of his large stock of material, as well as Dr. A. O. J. Kelly," seems to require explanation. Why should he thank Dr. Deaver for the use of Dr. Kelly? It sounds like a form of peonage. And why should the "inspired declaration of the great apostle"—"Others have labored,

ye have entered into their labors"—be regarded as appropriate for a work on the appendix? If it had been a text-book of obstetrics its applicability would be obvious.

Pleasantries apart, there is little to say of the book except in praise. The former and larger work is so well known that an extended review of this resume does not seem indicated. The history of the diseases and surgery of the appendix is accurate and interesting. The anatomy is well described and well illustrated. The chapter on the physiology of the appendix is almost as brief—and for much the same reason—as the celebrated chapter on snakes in Ireland. The author's attitude about it is, however, highly to be commended as displaying the true scientific spirit. Most of us—including the reviewer—have written of the appendix as "merely vestigial" and as "functionless," but it is apparent on reflection that such statements may or may not be true, and should not be unqualified. Dr. Kelly, beginning with the question: "Has the vermiform appendix any function?" answers it as follows: The argument from analogy for a special function may be briefly stated thus: In the first place, the functions of a number of organs heretofore considered functionless, as the thyroid, thymus, and suprarenal glands, have proved of such inestimable value—nay, so necessary to normal life—that the scientific physiologist will do well to reserve his judgment in regard to the appendix, and occupy an attitude of expectant observation. Again, as in the upper part of the digestive tract special secretions are poured in from such important organs as the liver and pancreas, so in the lower may we not expect, after the sudden transition from ileum to colon, to find some analogous organ or organs profoundly modifying the food under its altered conditions? He adds that we await the final test of demonstration. The bacteriology and pathology of the appendix are admirably summarized, and in this latter section—as, indeed, throughout—the illustrations are to be especially commended.

The chapter on the clinical history of appendicitis includes a consideration of the most common of the complications and sequels, and could scarcely be improved upon. In the chapter on diagnosis it is stated that "in some obscure cases a valuable aid in examining the patient, described by B. McMonagle (personal communication), is the pain elicited by holding the fingers firmly over the abnormal site of the appendix and requiring the patient to contract the right psoas muscle by flexing the lower limb, held rigid at the knee, on the body." This seems itself obscure. How is the "abnormal site" determined in "obscure cases" before this procedure is adopted?

The differential diagnosis is comprehensive and instructive. In the chapter on appendicitis and typhoid fever the author quotes with approval the opinion of J. B. Murphy, who says: "It is my opinion that typhoid appendicitis should not be operated upon, unless there is a perforation. All my cases recover, those operated and not operated. At the same time, I feel that operation should not

be performed except in special cases." Later (page 256) the author says: "Typhoid appendicitis rarely calls for operation, and it is always a lamentable misfortune to subject such a patient to an unnecessary surgical operation with a long, wearing illness before him." If this were the accepted rule of the profession—as it is coming to be—many lives would doubtless be saved. Appendicitis in childhood receives full consideration, and the excellent paper of Meriel on appendicitis occurring after the fiftieth year is fully summarized. Typhlitis—the despised and rejected—is once more recognized as a distinct pathological entity, though it is rare and far from being equally distinct clinically. The chapters on treatment are full of valuable advice. There are few surgeons who will not subscribe to the author's two concluding "aphorisms:" "It is better to operate occasionally in error in a doubtful case than to wait too long for more positive signs and to lose a life now and then. No patient is ever killed by a skilful early operation. Many die for want of one." "However, this facility with which an operation can be done must not be used as an excuse for slipshod and unnecessary operations."

The advice to the general practitioner who does not often do surgical operations that he would do better not to operate upon an appendicitis case when a satisfactory surgeon cannot be found, unless there is a well-defined mass in the right iliac fossa, with heat and tenderness—evidence of abscess formation—is certainly sound, as are the subsequent easily understood and excellently simplified directions for this procedure in case operation is unavoidable.

The articles on the operative treatment and on the postoperative sequels embody Dr. Kelly's well-known views, which are substantially unchanged. The chapters on appendicitis in gynecology and in obstetrics, on newgrowths of the appendix, on its hernias, and on the medicolegal aspects of appendicitis, are up to the high standard of the rest of the book, which contains more valuable information on the subjects treated of than is to be found in the same number of pages elsewhere. A little looseness in writing—or in the condensation of the material of the larger book—is occasionally noticeable, *e. g.*, "There are no symptoms nor combination of symptoms" (p. 154), but, as a rule, the information given is presented in an attractive and interesting manner. J. W. W.

THE PANCREAS. ITS SURGERY AND PATHOLOGY. By A. W. MAYO ROBSON, D.Sc. (Leeds), F.R.C.S. (Eng.), London, and P. J. CAMMIDGE, M.B. (Lond.), D.P.H. (Camb.), London. Pp. 546; 175 illustrations. Philadelphia and London: W. B. Saunders Company, 1908.

THE various diseases of the pancreas, inflammatory, cystic, and neoplastic, have been well understood for many years, and but little has been added to the work of such pioneers as Fitz and Senn, for

instance. The etiology of acute pancreatitis, the relation of the islands of Langerhans to diabetes, and the development of chemical tests whereby disturbance of the function of the pancreas may be detected, are instances, however, of details in which advances have been made. The treatment of pancreatic disease by surgical measures is of more recent date, and to the senior author of this monograph is due great credit for his persistent energy and pioneer work, especially in the treatment of chronic pancreatitis.

The first one-half of this large volume (Chapters I to X) is devoted to the anatomy, physiology, and pathology of the pancreas, and includes a chapter upon fat necrosis. These subjects are well handled and complete, most of the matter relating to the so-called chemical pathology of the gland being devoted to a consideration of the changes in the urine and feces, with especial reference to the well-known test of Cammidge, the junior author of this monograph. The authors state that they "never rely upon the 'pancreatic reaction' alone in making a diagnosis of pancreatitis or malignant disease of the pancreas, but always take into account the results of a complete analysis of the urine and a chemical examination of the feces, as well as the clinical symptoms." The improved or *C* reaction is intended to supersede the methods published previously by Cammidge; its technique is long and complicated, and in its present form it would seem to be available only to those who are versed in chemical methods.

Chapter XI is devoted to the subject of pancreatic diabetes, and although it is suggested that such may be prevented by operating early upon cases of pancreatitis and gallstones, it hardly seems worth while to devote over forty pages to it in a book of this character. Chapter XII, while mostly a repetition of the subsequent text, groups all of the symptoms and tests which may be of use in diagnosis, the chemical tests occupying much more space than the symptoms. The injuries and inflammatory affections are considered next (Chapter XIII to XVI), and, with the exception of some padding with case reports, present an excellent and complete description of the symptoms and treatment of these affections. The authors urge early operation for the acute cases, and advise rapidity in the execution of the operation. In chronic interstitial pancreatitis cholecystenterostomy is preferred, and the authors believe that "only in case of absence or contraction of the gall-bladder, or in case of unusual difficulties from adhesions or from the serious condition of the patient should cholecystotomy be done." The remainder of the text is devoted to pancreatic calculi, cysts, and tumors, which are discussed in a brief but satisfactory manner.

The illustrations generally are good, although some, Figs. 120, 150, and 174, for instance, could not exist without their legends. An index of authors is appended, and a bibliography at the end of each chapter adds materially to the value of the volume. G. P. M.

CLINICAL DIAGNOSIS. A Text-book of Clinical Microscopy and Clinical Chemistry. By CHARLES P. EMERSON, M.D., late Resident Physician, Johns Hopkins Hospital, Baltimore. Second edition; pp. 686; 126 illustrations. Philadelphia and London: J. B. Lippincott Company, 1909.

ESSENTIALS OF MEDICINE. By CHARLES P. EMERSON, M.D. Pp. 383; 117 illustrations. Philadelphia and London: J. B. Lippincott Company, 1909.

THE first edition of Emerson's *Clinical Diagnosis* was noted in these columns shortly after its publication, and its commendable features were pointed out. In its second edition, the book has been much revised and considerably enlarged; the author states that he has re-written fully one-half of it, and that he has incorporated much new matter, notably the bacteriology of the sputum and the urine, and a number of new illustrations. It is not necessary to speak in detail of a book now so well known and widely used; it suffices to chronicle the publication of a new edition and to say that the book fully meets the requirements of clinical laboratory diagnosis.

In the mean time, Dr. Emerson has prepared another and smaller book, which comprehensively is designed as a text-book for students beginning a medical course, for nurses, and for all others interested in the care of the sick. The avowed purpose of the book attracts some attention, since it is no mean task to encompass in a small volume the special requirements of the three classes of persons mentioned. Personal inclinations commonly influence judgments regarding what properly should be included within essentials, so that opinions concerning the value of the book necessarily must vary; but bearing in mind the task imposed upon himself by the author, one may say that the book contains many facts clearly and succinctly stated—it is true, in a more or less popular phraseology, but well adapted to ready assimilation by those that know little of the subject. In addition, however, the book may be read with interest and profit by those who have passed beyond the initial stage of learning. A. K.

A TEXT-BOOK OF OPERATIVE SURGERY. By WARREN STONE BICKHAM, M.D., Phar.M., Junior Surgeon to the Touro Hospital, New Orleans; late Assistant Instructor in Operative Surgery in the College of Physicians and Surgeons, Columbia University, New York. Third edition; pp. 1206; 854 illustrations. Philadelphia and London: W. B. Saunders Co., 1908.

THE first edition of this text-book of operative surgery appeared in 1903, and was followed by the second edition in 1904, now nearly five years ago. As might be expected, it has been necessary to add

much new matter, owing to the development of new fields of operative surgery during this period. The volume is larger by nearly 200 pages, and contains about 300 more illustrations than the second edition. The plan of the volume remains unchanged, and the same criticism can be made now that was made by the reviewer of the first edition, namely, that the constant repetitions required in the effort to make the description of each operation complete in itself add unnecessarily to the size of the book, and detract from its value as a text-book, though perhaps rendering it more useful as a book of reference. The failure to discuss the indications and contra-indications for operation, or the dangers, results, and complications of the operations described, also makes it less useful as a text-book than is a volume which includes a certain amount of surgical therapeutics. The amount of surgical anatomy included is minimal. But as the author makes no pretence to give more than the technique of the operations, these shortcomings cannot be considered as justifying adverse criticism; and to urge the inclusion of more matter would be injudicious, the volume being excessively bulky as it is.

Plastic surgery is still excluded, though it surely forms more a part of general surgery than such procedures as osteoplastic resection of the spine, an operation never very widely employed, and now, very properly, we believe, in disrepute: yet to this one subject nearly thirteen pages are devoted. The section on amputations is especially complete. Orthopedic surgery is slighted, as are gynecological operations. While the operations on bones in general are sufficiently described, bone wax is not even mentioned. The accounts of prostatectomy are antiquated, the author apparently having failed to grasp the essential points of the technique of suprapubic prostatectomy as practised by Freyer, and of the perineal operations developed by Albarran, Hartmann, and Proust. Young's method of "conservative perineal prostatectomy" is not mentioned. Dr. Bickham states that "other" operations on the prostate than those described have been omitted as belonging more especially to genito-urinary surgery; but to relegate all the best operations to the realm of specialism, and to permit the general surgeon to attempt only those which are truly blind, bloody, and barbarous, is to enunciate a principle in surgery which we believe to be thoroughly erroneous.

The operations on the gall-bladder are incompletely and inadequately described, and the operations of gastrostomy known by the names of Marwedel and Ssbanajew-Frank (not Franck), might well have been omitted. The account of perineal cystotomy (lateral lithotomy) is hazy, and the nomenclature of perineal operations on the male is in general confused, the old operation of "perineal section" (the "London" operation) being described as Wheelhouse's, merely because the use of Wheelhouse's staff is advised; and Cock's operation is called perineal section. The illustrations are very clear, even semidiagrammatic; but they sometimes will give a decidedly

false impression to the student, and not seldom are lacking in those minor details which count for so much in bringing an operation to a satisfactory conclusion. If another edition shall be called for, we will hope to see much condensation of essential matter, and to find many procedures seldom seen but in print totally excluded from the volume. A. P. C. A.

A Text-book of Special Pathology. By J. Martin Beattie, M.D., Professor of Pathology and Bacteriology in the University of Sheffield, and W. E. Carnegie Dickson, M.D., Lecturer on Pathological Bacteriology in the University of Edinburgh. Pp. 599; 191 illustrations. Philadelphia: P. Blakiston's Son & Co., 1909.

As in their *Text-book of General Pathology*, noticed in these columns several months ago, Drs. Beattie and Dickson, in their *Text-book of Special Pathology*, state that if any excuse were necessary for adding another to the many text-books of pathology, it would be found in the fact that the present volume is based on the teaching of the Edinburgh school, and that some fundamental points, which have been taught at Edinburgh for years, have not elsewhere received sufficient attention. Such, perhaps excusable, insularity is not the best incentive to the preparation of a scientific treatise and must be ignored in estimating the value of the book—which should be judged solely by its intrinsic merits. Thus judged, the book is found to contain the main facts of special pathology, clearly and concisely, in some places somewhat dogmatically stated. Special emphasis has been laid upon gross morbid anatomy—in which the book is relatively strong; but microscopic anatomy is rather neglected and there is little or no discussion of pathogenesis. There is some reason for excluding diseases of the eye and the ear; but the omission of all reference to the female genitalia, while including the mammary gland and the male genitalia, engenders the suspicion that perhaps *ennui* overtook the authors. What there is in the book is very good—the making of a really good book. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,

AND

W. S. THAYER, M.D.,

PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

A Clinical Procedure for the Recognition of Sugar in the Urine with Orthonitrophenylpropiolic Acid as an Indicator.—BOTTU (*Soc. de biologie*, 1909, lxvi, 972) describes the method of using orthonitrophenylpropiolic acid as an indicator of sugar in the urine. He says that it is superior in sensitiveness and precision to the numerous copper or alkaline bismuth solutions in general use. The reagent is made by mixing 5 c.c. of fresh 10 per cent. sodium hydroxide with 3.5 grams of pure orthonitrophenylpropiolic acid powder and adding distilled water to make up to a liter. Its employment consists in adding 1 c.c. of urine to 8 c.c. of the reagent in a test-tube, and heating the upper layers. As soon as it comes to a boil, add drop by drop another cubic centimeter of urine. Then heat again. If the urine contains glucose, there will appear a blue color with more or less precipitation of small particles of indigo blue. If the reduction occurs spontaneously before the second boiling, the glucose content can be estimated to be 10 grams per liter or more. The reaction is trustworthy, very easy, and sensitive.

Statistics of Gonorrhœal Arthritis.—In the course of a fifteen-year practice, among 820 cases of gonorrhœa, JORDAN (*St. Petersburger med. Woch.*, 1909, xxxiv, 59) has observed eighteen cases of gonorrhœal arthritis. This is a frequency of 2.1 per cent., and agrees well with the figures of other reports. The time of appearance of the arthritis has varied. Five cases occurred in five to seven days after the onset of the urethritis; 2 cases, in the course of the first two months; 11 cases, in the chronic stage. However, in most reports the figures of early occurrence are not so reliable, inasmuch as they depend solely upon the statements

of the patients as to the onset of the urethritis. One thing is certain, that is, arthritis may be an early complication of specific urethritis. As predisposing causes of the arthritis, exposure to cold, trauma of the affected joints, erection and trauma of the urethra must be considered. In addition, an individual predisposition to gonorrhœal arthritis is illustrated by two cases already so affected in previous attacks of urethritis. The age of the patient is no factor in the frequency of the complication. A single joint was affected in 7 of the 18 cases, 2 joints in 5 cases, 3 joints in 2, and 4 in 4 cases. Of the single joints, the knee was involved twelve times, the ankle eight times, the wrist four times, the elbow twice, the shoulder and the hip each once. Six cases showed simple hydrops, 11 serofibrinous exudate, and only 1 case phlegmonous. The fever was mild and intermittent. As to prognosis, 9 cases came to restitutio ad integrum. In 5 slight stiffness remained with swelling of a joint. Two had outspoken ankylosis and 2 refused treatment. The author adds nothing from his own experience as to the results obtained with Bier's hyperemia and the newer methods of treatment.

The Anatomical and Clinical Relations of the Sphenopalatine (Meckel's) Ganglion to the Nose and its Accessory Sinuses.—SLUDER (*New York Med. Jour.*, 1909, xc, 293) calls attention to the intimate relation of Meckel's ganglion to the walls of the accessory sinuses of the nose and the common wall of the nose itself. It lies close to the top of the pterygopalatine fossa just below the sphenoidal sinus. The sphenopalatine foramen is accurately placed just posterior to and immediately below the posterior tip of the middle turbinate bone; and the ganglion is close to the plane of this foramen, from 2 to 9 mm. from the general membrane of the nose. With such intimate anatomical association, clinical manifestations from the extension of inflammation or its products would seem of almost necessary occurrence. Sluder has observed characteristic disturbances follow postethmoidal and sphenoidal inflammations which cannot be explained otherwise than by assuming that Meckel's ganglion had become involved by extension. This has also occurred from the nose proper, but never, so far as known, from the maxillary sinus. In such cases of grippe origin, the pain has persisted, neuralgic in nature. The typical picture, as drawn from 47 cases, is of pain which begins at the root of the nose, extends downward over the maxilla, and backward on the mastoid, to become severest about 5 cm. posteriorly to its tip. Then extending backward, it takes in the entire occiput, and down into the neck, shoulder blade, shoulder, and sometimes the axilla. With the severest attacks it extends into the arm, forearm, hand, and fingers. It rarely invades the upper part of the head. It may extend into the brow or the zygoma. Concurrent salivation, a "metalic" taste, earache, toothache, and pain behind the eye have been noted. Sluder has applied cocaine solution to the nasal mucous membrane overlying the ganglion with success, and has injected alcohol directly into the ganglion, aborting attacks successfully.

A New Method of Recognizing Ulcers of the Upper Alimentary Tract and Determining their Localization.—EINHORN (*Berl. klin Woch.*, 1909, xlvi, 742) has devised a method for recognizing positively the presence

of ulcers in the upper digestive tract, and especially their localization. At nine in the evening the patient swallows in a gelatin capsule the so-called duodenal bucket devised by the author, to which a thread of twisted silk is made fast. This thread can reach from the lips 75 cm. in the alimentary canal. The next morning at seven or eight o'clock, while the patient is fasting, the apparatus is withdrawn. In cases of ulcer, where the thread comes into contact with the latter there is a brown or black stain on that exact portion. The distance of this from the lips gives the situation of the ulcer. With this "thread-impregnation test," ulcers in the œsophagus, cardia, smaller curvature of the stomach, pylorus, and duodenum can be recognized, whereas it is not so practical for those in the greater curvature and fundus.

Cutaneous Pigmentation as an Incomplete Form of von Recklinghausen's Disease.—F. P. WEBER (*Brit. Jour. Dermat.*, 1909, xxi, 49), in 1906, reported the case of a fifteen-year-old girl in whom the skin of the trunk and neck was mottled with numerous pigmented areas varying in size and shape. The earliest pigmentation was at the back of the neck, noted by the mother when the patient was only eighteen months old. Since then it had gradually developed, and relatively faint spots and patches had recently appeared on the extremities. The variety in color and form of the areas of pigmentation and their situation (mainly on the trunk) made it seem most probable that the case was an incomplete form of von Recklinghausen's disease. Only one small, flaccid, molluscous tumor was found on the lower back. In 1908 the diagnosis was confirmed by the formation of several other molluscous-like tumors in various parts of the skin. The pigmentation was darker and new brown spots had appeared. Though the term "von Recklinghausen's disease" should be confined to cases showing, (1) obvious neurofibromas in connection with nerve trunks, (2) molluscous tumors of the skin, and (3) cutaneous pigmentation, yet Weber feels that incomplete or anomalous forms certainly occur in which one or even two of this triad of morbid features may be wanting. He would classify these forms as follows: (1) Cases of plexiform neuromas unaccompanied by molluscous skin-tumors, with or without cutaneous pigmentation. (2) Cases of multiple molluscous tumors unaccompanied by any obvious neurofibromatosis of the nerve trunks, with or without any decided pigmentation. (3) Cases of pigmentation without neurofibromas of nerve trunks or cutaneous neurofibromas. (4) Anomalous cases of neurofibromatosis complicated by the co-existence of bony or epidermic changes.

Abolition of the Corneal Reflex as a Diagnostic Sign of Hemiplegia in Coma.—MILIAU (*Progrès méd.*, 1909, xviii, 229) emphasizes the importance of the loss of the corneal reflex in differentiating a hemiplegia during coma. When the cornea is touched, the lids are normally contracted at once and the eye closed. While an individual is in coma, if he has a hemiplegia, this energetic reflex is abolished. This occurs on the side of the paralysis, and is absolutely constant. Miliau considers it valuable evidence and has found it most useful. In crossed paralysis, in bulbar lesions, the loss is on the side of the facial paralysis and

opposite to the paralyzed limbs. In Jacksonian epilepsy the reflex is lost on the side of the convulsive seizures, and on both sides when the convulsions become general. This is particularly useful in distinguishing hysterical convulsions from true epilepsy. If the comatose condition is due to an intoxication, the reflex is absent bilaterally. Chloroform narcosis is an example of this. In uremia, however, reservations must be made, inasmuch as this intoxication is occasionally accompanied by partial paralysis dependent upon cerebral lesions, either toxic or due to œdema.

The Clinical Value of Antitrypsin Determinations in the Blood.—JACOB (*Münch. med Woch.*, 1909, lvi, 1361) has studied the antitryptic content of the blood in various diseases, his material numbering about 100 cases. Fuld's method was employed in the experiments. His study leads Jacob to conclude that an increase of antitryptic ferment of the blood serum occurs in a relatively large number of diseases. It is practically constant in severe cachexia, but is also very frequently present in all diseases associated with changes in the white blood cells. Finally, it may be observed in a number of cases in which neither of these factors is present. The occurrence and the cause of the reaction are still quite obscure; a positive result is obtained in such a variety of diseased conditions that no diagnostic significance can be attached to it.

A Reaction in the Umbilical Blood of Newborn Infants: A Contribution to the Study of Much's Psychoreaction.—BAUER (*Münch. med. Woch.*, 1909, lvi, 1367) has studied the reaction described by Much in the blood of patients suffering with dementia præcox, etc., in which a positive result is indicated by inhibition of cobra hemolysis after the addition of the patient's serum to the hemolytic material. The author finds that serum obtained from the umbilical cord of the newborn gave a positive result in practically all of 17 cases. The blood serum of 16 older children and adults in no case gave a positive reaction, while of 9 sucklings examined, only one was positive. The results obtained by the author indicate that the reaction is not specific, and the name "psychoreaction" should not be applied to it. The author's investigations are being continued.

Vaccine Bodies.—CZAPLEWSKI (*Deut. med. Woch.*, 1909, xxxv, 1152) obtained human, somewhat "purulent" lymph from a vaccinated child for study. Cultures on various media showed the lymph to be free from bacteria; stains also failed to demonstrate any bacteria, but the author considered the stains unsatisfactory, for no leukocyte nuclei were found in this "purulent" material. (Gram-Weigert with carbol-fuchsin counterstain, gentian violet, carbol-fuchsin and Giemsa stains were tried). By a special modification of the staining technique the author obtained very unexpected objects, which he later demonstrated also in vaccine lymph from man, the calf, and the rabbit. Further study revealed new forms which reminded the author, both in appearance and staining reaction, of the cycles of certain protozoa, especially coccidia, hemosporidia, and microsporidia. The author believes he has observed both asexual (schizogomie) and sexual (sporogonia) cycles, corresponding to

the double cycles of coccidia and malarial parasites. The asexual and sexual forms are described in various stages. In a detailed paper, soon to appear (*Zentrabl. für Bakt.*), the technique will be fully described and microphotographs published.

The Treatment of Typhoid Bacillus Carriers.—LIEFMANN (*Münch. med. Woch.*, 1909, lvi, 509) discusses the problem of the disposal of chronic typhoid bacillus carriers who are themselves in good health, but a source of danger to others. It is important naturally to render them harmless to the community, and yet the many researches for this purpose have been without results. But it seemed to Liefmann that good results might be obtained with the Bulgarian sour milk, the so-called Goghurt, which is such a favorable medium for the growth of intestinal flora. He fed this to three bacillus carriers for a long period, taking care that the diet be not so rich in proteins as to interfere with a strong intestinal putrefaction. After seven weeks typhoid bacilli were found in the stools of one of the individuals, but never again since, although search was kept up diligently for a period of eleven weeks from the beginning of the treatment.

The Rare Forms of Cyanosis: Polycythemia, Methemoglobinemia, and Sulph-hemoglobinemia.—CLARKE (*Med. Record*, 1909, lxxvi, 143) describes the commoner forms of cyanosis due to imperfect aëration of the blood, as in heart disease, pneumonia, pleurisy, emphysema, and tuberculosis. Polycythemia is the form occurring in conjunction with a high red blood count. But the rarest of all forms is that produced by an alteration of the blood pigment itself. If this abnormal pigment is methemoglobin, the condition may be due to drug poisoning (acetanilide, phenacetin, sulphonal, trional), or auto-intoxication by the absorption of nitrites from the intestines in cases of chronic diarrhœa. If the pigment is sulph-hemoglobin, it is probably associated with chronic constipation, and is the result of the hyperformation or hyperabsorption of H_2S or the presence in the blood of an abnormal reducing agent acting with a small trace of H_2S. The methemoglobin cases improve as the enteritis is cured, and the sulph-hemoglobin cases recover if the bowels are kept open.

The Ipecac Treatment of Amœbic Dysentery.—DOCK (*New York Med. Jour.*, 1909, xc, 49) emphasizes the use of ipecac in chronic amœbic dysentery, in which he has had remarkably good results. From experiments, he suggests a direct destructive action of the drug. In addition there is a cholagogue and sometimes a purgative action. Clinically, in cases treated with ipecac a cessation of amœboid motion of the parasites occurs in a few hours. In one to three days they disappear completely. In the same time the stools lose their dysenteric character. Mucus and blood, later pus, disappear, and the stools resemble those of mild enteritis or even become formed. The effect upon the other protozoa in the stools varies. The patient should always be kept in bed. The ipecac is sometimes depressing. With the idea of getting the remedy into as close contact as possible with the parasites, there should be a preliminary pugation with salts. All food should be stopped while the ipecac is being taken, and only liquids

used after it. The dose of ipecac varies with the severity of the case, from 30 to 60 grains being given at first, and then 20 to 40 grains twice a day, for three days. This is less troublesome and just as efficient as smaller doses at frequent intervals. Coating the pill with salol or keratin prevents solution in the stomach and attendant emetic action. Salivation is occasionally produced. Griping may follow, but is easily controlled with atropine.

Chronic Cholecystitis as a Cause of Myocardial Incompetence.—BABCOCK (*Jour. Amer. Med. Assoc.*, 1909, lii, 1904) reports 13 cases in which the effect of cholecystitis is seen in myocardial incompetency; 5 of these cases suffered with dilatation, arrhythmia, and feebleness of the heart's action plus murmurs: in short, subjective and objective symptoms of serious heart disease. Two cases had attacks of pain that had been called angina pectoris or were of an anginoid character. Evidences of myocardial inadequacy followed the attacks. A group of 3 cases had intermittence of the pulse of long standing and very intractable. Two cases of valvular disease suffered a decompensation due to outspoken attacks of hepatic colic. The explanation of the effect of the gall-bladder disease upon the heart is difficult. Several theories may be advanced: (1) The chronic myocarditis may be the result of the chronic gall-bladder infection—the circulation of bacteria and their toxins. The acute myocarditis due to an acute cholecystitis is well known. (2) The bile constituents have a depressing effect upon the myocardium. Stewart and King have proved that biliverdin is toxic to heart muscle and that bile salts increase the blood pressure. (3) Inasmuch as the splanchnics are the regulators of the circulation, their disturbance may prove an added factor by unfavorably affecting an already weakened heart and so leading to its dilatation and incompetence. (4) The disastrous effect of biliary colic upon the heart is probably a reflex result of stimulation of the filaments of the pneumogastric distributed to the wall of the gall-bladder. It is probable that there must be a predisposing cause residing in the heart muscle, a chronic myocarditis, in consequence of which the heart is unfavorably affected by influences which a healthy myocardium would be able to resist.

Meat as a Source of Infection in Tuberculosis.—LITTLEJOHN (*Practitioner*, 1909, lxxxii, 843) states that, to the community at large, the risk of contracting tuberculosis by eating the meat of tuberculous animals is not so great as generally believed. Imperfect inspection makes the risk greater than it should be, a risk which concerns especially those who buy cheap meat and eat such commonly infected organs as the lungs, udder, and mesenteries. The relative infrequency of tuberculosis in all but cattle and pigs makes them the only food animals at all dangerous to man. The flesh of tuberculous animals is not necessarily infectious, and when so, in many instances, it is due to secondary contamination by means of the knife, butcher's cloth, and dressing. Cooking is sufficient ordinarily to destroy tubercle bacilli on the outside of a roll or joint, but not in the centre. Finally, digestion, salting, or smoking have little or no disinfecting effect. However, the rarity of tuberculous foci in the flesh, even in advanced cases, makes ordinary cooking efficient if the thick rolls and joints are avoided and the meat is well done.

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY HOSPITAL,

AND

T. TURNER THOMAS, M.D.,

ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL, AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

The Surgical Treatment of Benign Affections of the Stomach, Especially of Gastric Ulcers and their Sequels, with Particular Reference to their Late Results.—BUSCH (*Archiv f. klin. Chir.*, 1909, xc, 1) says that most German surgeons are agreed that gastro-enterostomy is the normal treatment for gastric ulcer, and that resection is justified only when there exists an indurated tumor, in which there is some suspicion of cancer. Kronlein considered that operation was absolutely indicated in the presence of pyloric stenosis, in atonic gastresia and gastroptosis, and in frequently recurring slight hemorrhages. Busch reviews 124 cases of benign affections of the stomach operated on by Körte, in which 129 operations were performed. Of 76 ulcer cases, 65 were completely free of trouble more than one year after operation, 8 were able to work with slight stomach disturbances, and in 2 cancer developed in the floor of the ulcer. In 85.5 per cent. of the cases in which long continued internal therapy had failed, a successful result was obtained after operation. In 12 resections the end-results were good in all, although in 2 cases another operation was necessary because of a recurrent stenosis. He concludes that for ulcer stenosis, as well as for ulcer persisting in spite of long-continued internal treatment, the posterior, retrocolic gastro-enterostomy, by means of a circular double row of sutures, is the operation of choice. Especially for an open ulcer it will be advisable to keep up, for several weeks after operation, treatment with food of reduced acidity, with later washing out of the stomach, and a careful selection of foods. Resection should be done when there is a suspicion of malignancy, or in callus ulcer, especially when they press upon neighboring organs, as the liver and pancreas. It should always be circular according to Riedel's suggestion. In many cases it will be doubtful whether radical resection or the less dangerous gastro-enterostomy should be done. The nervous diseases of the stomach should not be operated on. There are, however, cases in which only an exploratory laparotomy will determine whether an ulcer exists or not.

The Value of Intravenous Adrenalin-Saline Solution in the Treatment of Peritonitis.—HEINECKE (*Archiv f. klin. Chir.*, 1909, xc, 102) says that the excellent results obtained by Heidenhain in peritonitis, by the intravenous infusion of adrenalin-saline solution, are remarkable, in view of the transitory influence of adrenalin in raising the blood pressure. No investigator has yet observed a lasting effect on the blood

pressure, even for hours, in animal experimentation; nor has anyone observed that its influence is exerted on the vasomotor centres, so that this theory must have a purely hypothetical basis. The disturbances depend upon a paralysis of the vasomotor centres. The intravenous infusion will raise the blood pressure in every stage of collapse in peritonitis, but only for a few minutes. Its effect is no greater on the blood pressure in collapse than in a normal condition. When the salt solution is added, the effect of the adrenalin will last somewhat longer, but this is probably due to the effect of the salt solution, not of the adrenalin. The influence of the latter must, therefore, be very limited, and the success of Heidenhain must have been due to the salt solution, the influence of which on the blood pressure is much less than that of adrenalin, but much more permanent. Its effect, however, is not constant. In those cases in which there has been a loss of much body fluid its effect is very marked, and may be life saving, but when this condition does not exist it is not very valuablee.

Operations for Recurrence in Trigeminal Neuralgia.—BÜDINGER (*Deut. Zeitsch. f. Chir.*, 1909, xcix, 164) says that the present views concerning the dangers to which the eye is exposed in extirpation of the Gasserian ganglion are too optimistic, and that it is desirable that some method be sought which may be substituted for it. He reports a case in which the neuralgia was confined to the left inferior dental and lingual nerves. There was continuous lacrymation from the left eye, but no other disturbances in it. The two nerves were exposed within the mouth near the entrance to the inferior dental canal, and a 1 cm. piece of each removed cranialward. About a half-year later the pain recurred and was soon as bad as ever, and the patient much depressed. About a year after the first operation the trunk of the inferior maxillary division of the fifth nerve was resected to the base of the skull, after a temporary resection of the zygoma. The attacks of pain were again relieved for nearly a year, when they began again and soon became of the old severe type. Extirpation of the ganglion was now proposed to the patient, and was declined because of the risk to the eye. About ten months after the second operation the base of the skull was exposed as in the second operation. Then the bone was chiselled away in an area about 1.5 cm. in diameter, from the lowest part of the temporal fossa. The medial border of the defect was about 1 cm. distant from the foramen ovale. The bridge of bone between the two was then chiselled away. The trunk of the inferior maxillary nerve in the region of the bony canal was thickened and club-shaped, and sent fine twigs into the old scar tissue. It was isolated, and by pressing outward the dura, was followed to the ganglion, cut through, and drawn out as a whole. The hemorrhage stopped after a short compression. A piece of the bone chiselled away was properly shaped so that it fitted exactly into the foramen ovale and was wedged into the foramen to prevent the regeneration of the nerve tissue. The external portion of the bone defect was left open, and the wound was closed in the usual way. The typical attacks of pain did not recur after this operation. The chief objects of the operation were: to open up the foramen ovale in order to remove the neuromatous formation from its narrow bony canal, to remove as

much as possible of the nerve trunk, and to close the canal with bone to prevent regeneration. The fact that there has been no recurrence up to the present time, three and one-half years after the last operation, shows its advantage over the other peripheral operations, and there is the possibility that the cure may be permanent.

Fracture of the Astragalus with Luxation of the Fragments.—MAUCLAIRE and PETTITEAU (*Archiv. gén. d. chir.*, 1909, iii, 575) say that these fractures do not include dislocations complicated by fracture, since the dislocation in such cases is the dominating condition, but those cases in which the fracture is the first condition to be noted, as when a displaced fragment is detected under the skin. These fractures are very rare. The displacement is due to indirect force, the traumatism inducing secondarily forced movements of the foot in which the bone is broken and the fragments displaced. When the fracture is anterior to the interosseous ligament uniting the astragalus to the os calcis, we have a fracture of the neck or a decapitation of the astragalus. In this case the posterior fragment is fixed by the ligament and retains its normal position. The anterior fragment is caught between the weight of the body above and the bones below, so that the anterior fragment is enucleated upward under the skin on the dorsum of the foot. When the fracture is posterior to the interosseous ligament, the body of the bone loses its strong attachment and is luxated. The line of fracture may be more complicated, so that there may be several fragments. In the treatment two objects are sought after—to place the foot in good position and to obtain good function. The reduction differs from that in a fracture of a long bone, in that the fragments are more or less removed from their original location and are maintained in their abnormal position by a ligamentous apparatus which is itself dislocated. In a number of the ten collected cases a non-operative reduction was attempted without success. Such a reduction may be successful rarely, but even then it cannot be maintained with the aid of compression, and vicious callus develops, interfering with the function of the tibiotarsal joint. The frequent communication with an important joint tends to defeat consolidation and to produce a concomitant arthritis. In three cases reduction was accomplished by operation, in all of which there was a transverse fracture of the neck. The joint was freely opened and the fragments coapted. The functional results appear to have been excellent. Astragalectomy has proved to be an excellent operation. With vigorous asepsis it is a benign operation, which leaves perfect functional results, except in running and jumping. In all the cases it gave good results, except in that of Frischen, which resulted fatally. This occurred previous to the period of antisepsis. A total astragalectomy is usually preferable to a partial. In a compound fracture an astragalectomy should be done as soon as possible.

The Technique of Intrathoracic Resection of the Œsophagus.—TIEGEL (*Zentrabl. f. Chir.*, 1909, xxxvi, 1009) says that the end-to-end union of the œsophagus to the stomach has certain advantages over the lateral anastomosis as recommended by Sauerbruch. It saves a valuable portion of the œsophagus which must be sacrificed for a lateral anasto-

mosis, in which it is also difficult to place the sutures. The union is also more secure than in the latter. The method suggested by Tiegel is based upon experimental work on dogs, and makes use of a modified Murphy button. This consists in giving to the male portion of the button a more expanded bell-like shape. An obturator is made to fit into it and to catch by a spring which may be loosened by light pressure of the finger. The female portion is exactly the same as in the Murphy button. After the cardiac portion of the stomach has been completely separated from the diaphragm and the vagi nerves from the œsophagus, the œsophagus is divided by a Paquelin cautery between two clamps, about 4 cm. above the stomach. After compressing the cardiac end by a piece of gauze the clamp is removed and the female portion of the button is introduced into this portion of the œsophagus and the clamp again applied. The button is then directed into a portion of the fundus of the stomach by the finger, and is held there by a compress or a loose ligature. If there is a tumor at the cardia the button must be introduced through a small incision, which must be immediately closed by a clamp or suture. The œsophagus is ligated below the tumor, and this, with the attached piece of œsophagus, is removed by a Paquelin cautery and the edges of the cardiac opening in the stomach closed by turning in the edges with sutures. Then a movable piece of stomach is drawn into a cone with tenaculum forceps, and a purse-string suture introduced around and about 1.5 cm. from the point of the cone, including the serous and muscular coats. The ends of this suture are left loose. The oral end of the œsophagus is then turned forward by the attached clamp and a similar purse string suture placed in it, a loop being left opposite the free ends. Then a compress is placed on the œsophagus above and the end of the same cut through close to the clamp. With the aid of the ends of the purse-string suture and the loop opposite, the male portion of the button is easily introduced with the obturator. The suture is then tied around the cylinder of the button. The female portion of the button lying loose in the stomach is now forced into the cone already prepared for it, and an incision is made in the point of the cone over the button and just large enough to permit the cylinder to be forced through. The obturator is now removed from the œsophageal portion of the button and the two portions pressed together and locked. The purse-string suture in the stomach wall is drawn upward so that it may be tied above the male portion without constricting the œsophagus. This brings the serous surface of the stomach against a considerable portion of the outside surface of the œsophagus, and makes a good closure of the line of union between the œsophagus and stomach.

Cancer of the Kidney; Extirpation; Cure.—PILLET (*Ann. d. mal. d. org. gén.-urin.*, 1909, ii, 987) removed a cancerous kidney about the size of the fœtal head. The incision was made along the crest of the ilium, turning upward toward the thorax and ending in the epigastric fossa. Although extensive, this incision makes a good exposure of renal tumors. The musculocutaneous flap was turned over on the abdomen and the twelfth rib resected. The tumor was then enucleated after ligating the venous plexuses uniting the tumor to its capsule. The inferior pole of the kidney was enucleated with difficulty, the promontory being

nearly reached before this was accomplished. The abundant hemorrhage was controlled by a tampon. The tumor being brought out of the wound, the ligatures were tied and reversed several times about the pedicle, the tumor extirpated, and definite hemostasis provided. Drainage and sutures were then introduced. The operative results were normal. The urine passed on the first day was 450 grams, on the second day 1200 grams, and on the third day 1500 grams. From the eighth to the tenth day, 2.5 liters, a true polyuria of convalescence was present. Microscopic examination showed the tumor to be an epithelioma after the type of a hypernephroma. Five weeks after operation the patient left the hospital and took up his normal life.

Palpation of the Ureter at the Superior Straight of the Pelvis.—TADDEI (*Ann. d. mal. d. org. gén.-urin.*, 1909, i, 904) says that the origin of the common iliac arteries corresponds to the umbilicus. The course of the common and external iliacs is, therefore, outlined on the abdomen by a line from the umbilicus to the femoral pulse under Poupart's ligament, or if the femoral pulse cannot be palpated, to a point 1 cm. internal to the middle of Poupart's ligament. The bowels should have been purged, and the patient should be in a comfortable position with relaxation of the abdominal muscles by flexion of the thigh on the abdomen and elevation of the head and upper part of the trunk. The patient should breathe regularly with the mouth open. The palm of the hand is placed on the line already traced. The hand will cover the greater part of the line which will run about under the middle of the palm, the tips of the fingers being directed toward the umbilicus and reaching to about 3 or 4 cm. from the umbilicus. By deep pressure the pulsation of the common iliac artery is sought. In a large number of cases this pulsation is easily perceived. The pulsation of the common and external iliacs can be distinguished from that of the mesenteric vessels over them by the difference in force determined by their caliber. After recognizing the iliac artery by the tip of the middle or ring finger already in place, the other hand is placed in a similar position under the first, and the tips of the same fingers of the second hand, just outside of those of the first, are made to sink into the abdomen until they perceive the pulsation of the iliac artery. This will then bring out the ureteral pain. The fingers of the first hand serve to recognize the pulsation of the iliac, and to maintain the depression of the abdominal wall. This makes more easy the finding of the ureter by the hand underneath. Under favorable circumstances, that is, with lax abdominal walls, empty intestine, and thickened ureter from periurethritis, the ureter can be palpated for several centimeters.

Jejunal and Gastrojejunal Ulcers following Gastrojejunostomy.—PATERSON (*Annals of Surgery*, 1909, l, 367) says that the risk of jejunal ulcer following gastrojejunostomy is probably under 2 per cent. At the present time this complication apparently occurs less frequently than formerly. Clinically there are two groups of cases: (1) Those in which perforation into the general peritoneal cavity ensues; (2) those in which general peritonitis is prevented by the formation of adhesions. Pathologically the cases may be classified as follows: (1) Ulcers of the jejunum;

(2) gastrojejunal ulcers, or ulcers at the site of the anastomosis. Jejunal ulcers are in some instances of infective origin. In these cases ulceration commences within a very short time after gastrojejunostomy, and usually the ulcers are multiple. In a large proportion of cases the ulcer is single, and is probably the result of the toxic action of HCl, which injures the cells of the mucous membrane so that they are digested by the intestinal juice. Possibly agents other than HCl may play a part in injuring the mucous membrane. Gastrojejunal ulcers are the direct result of the wound made in effecting the anastomosis, and their persistence is probably the result of hyperacidity of the gastric juice. Closure of a gastrojejunostomy opening is the consequence of cicatrization of a gastrojejunal ulcer. It is more likely to occur when the pylorus is patent, not because of the patency of the pylorus, but because in such cases hyperacidity is usually marked. Any procedure or disease which diminishes the amount of bile and pancreatic juice in the jejunum, favors the occurrence of jejunal and gastrojejunal ulcers. For this reason operations of the "Y" type and entero-anastomosis are inadvisable, at any rate in cases in which free HCl is present in the gastric contents, as after these procedures the anastomosis and a portion of the jejunum are deprived of the protective influence of the alkaline bile and pancreatic juice. The reason that ulceration has followed the anterior operation more frequently than the posterior operation with a loop, is probably that in former times the anterior operation was more frequently performed. As no instance of ulcer after the posterior, no-loop operation has yet been recorded, we must for the present assume that its occurrence after this type of operation is less likely. It is possible, however, that this immunity is partly the result of improvement in technique and in the after-treatment of gastric operations in general. In cases in which perforation into the general peritoneal cavity occurs, immediate laparotomy offers the only chance of saving the patient's life. Inasmuch as there is some evidence that jejunal and gastrojejunal ulcers may heal, an operation should not be performed in the chronic cases until after a thorough trial of medical treatment. Even when surgical intervention is necessary, an attempt should be made to diminish hyperacidity, if this be present. Our aim should be to prevent the occurence of the complication of gastrojejunostomy. Preventive treatment consists, in (1) careful and appropriate surgical technique, and (2) prolonged after treatment. Lastly, every case of recrudescent pain of constant character after gastrojejunostomy, especially when associated with hyperacidity or hypersecretion, should be regarded as a case of potential ulcer, and treated accordingly.

THERAPEUTICS.

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF APPLIED THERAPEUTICS IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

Lactic Acid as an Agent to Reduce Intestinal Fermentation.—HEINEMANN (*Jour. Amer. Med. Assoc.*, 1909, lii, 372) says that the usefulness of lactic acid or lactic ferments as curative agents for intestinal fermentation is still problematical. Much clinical evidence has accumulated since Metchnikoff recommended the use of Bacillus bulgaricus. This bacillus digests some of the casein of the milk and also decomposes some of the fats, so that the taste of the milk becomes disagreeable. Metchnikoff advised the addition of the paralactic bacillus (Streptococcus lacticus) in order to obviate this difficulty. Heinemann thinks that the association of the two organisms leaves doubt as to which one is responsible for the beneficial effects claimed. Since Metchnikoff published his work, many other lactic acid producing organisms have been advised by different observers. Heinemann also believes that the experiments which have been reported in regard to finding the Bulgarian bacillus in the feces are incomplete in so far as no statements are made in regard to the number of the bacilli found in relation to other intestinal bacteria. Recently Herter and Kendall have investigated the fate of Bacillus bulgaricus in a monkey. They failed to establish the preponderance of the Bulgarian bacillus in the ileocecal region. They found that the number of these bacilli was relatively smaller in the large intestine. Their assumption is that the introduction of lactic acid bacilli reinforce the enfeebled action of the normal intestinal lactic acid bacilli against pathological varieties of bacteria. Heinemann believes that the question of the usefulness of bacteria in the digestive tract is a debatable one—although most authorities seem to agree that the production of acids in the colon is of value for the inhibition of the growth of putrefactive bacteria. However, Heinemann says that it has not been shown that lactic acid is produced in any considerable quantity by the presence of lactic acid bacteria in the intestine. It is true, though, that clinical evidence has shown that sour milk and pure cultures of lactic acid bacilli in connection with a diet in which milk is a prominent feature are of value for inhibiting intestinal putrefaction. Heinemann tested many of the commercial preparations for the artificial souring of milk. Samples were examined bacteriologically, their lactic acid forming properties were tested, and observations made with regard to the palatability of the artificial product. Ordinary sour milk, buttermilk, and the effect of the commercial preparations on pasteurized and boiled milk were also tested. Heinemann's experiments showed that the claims for the artificial souring of milk are not justified. Furthermore, it is largely questionable whether artificially soured milk possesses any therapeutic advantages over milk naturally

soured. He thinks that Streptococcus lacticus, the organism responsible for the natural souring of milk, produces about the right palatability. When milk has solidified from the coagulation of the casein brought about by Streptococcus lacticus it contains these bacteria in almost pure culture. Milk which has reached this stage is eaten by many people as a delicacy. Raw certified milk is preferable to boiled milk for use in the souring process, as boiling alters the taste. However, he thinks it better to boil or pasteurize the milk if certified milk cannot be obtained. If this is deemed necessary, an artificial starter must be used. Any of the commercial preparations serve for this purpose—lacto-bacilline, fermenlactyl, kefil, yoghurt, or lactone. A small amount of the artificially soured milk may be used for subsequent inoculations. Metchnikoff believes that the presence of yeasts is detrimental, yet all of the commercial preparations save one contained yeasts in large numbers. Yoghurt of the Bulgarians, kefir, koumyss, leben, and all the other fermented milk beverages contain alcohol-forming yeasts. Heinemann says that the question of taste is of considerable importance, and believes that the taste produced by the activity of Streptococcus lacticus with the addition of a yeast is without doubt the most preferable.

The Treatment of Obesity.—Von Noorden (*Med. Klinik*, 1909, i, 1) distinguishes two main forms of obesity. One form is due to overfeeding in proportion to exercise, and the other is due to deficiency of thyroid function. In the first form the power of oxidation is normal, but excessive eating or lack of exercise, or a combination of both, causes obesity. In the second form the power of oxidation is reduced, though the amount of exercise may be relatively normal. The influence of the thyroid may be primary or secondary. In the secondary forms the deficient oxidation may be due to an inhibitory action upon the thyroid secretion by diseases of other organs, such as the pancreas, ovaries, hypophysis cerebri, suprarenals, or thymus. Von Noorden divides cases of obesity into these two main classes, in order to facilitate their treatment. He treats the first class by the general principles of the usual obesity cure, which is a diet of low caloric value with an abundant supply of albumin. Exercise is a valuable adjunct, and oxidation may be increased by the withdrawal of warmth. If dietetic measures fail after a fair trial, he believes that thyroid insufficiency should be suspected. He thinks that a careful and systematic thyroid treatment is much less taxing to the patient than the ordinary dietetic treatment, especially the "starvation cases." He allows his patients on thyroid treatment a liberal diet, especially of proteins. He says that dietetic measures and exercise have no effect upon cases of obesity of thyroid origin except that of weakening the patient.

The Dietetic Treatment of Obesity.—Strauss (*Therap. Monat.*, 1909, i, 17) reviews the dietetic treatment of obesity as advocated by many different observers. He thinks that the Karell "milk cure," with its various modifications, and the Rosenfeld "potato cure" are practically starvation diets. They are only applicable to those patients who can be kept in bed or at rest. These diets are especially low in

protein, so that the nitrogen equilibrium is not maintained. Consequently part of the loss of weight in such cases is due to the loss of protein substances from the body. He says that it is unfortunate that the ease of their application should be one of the chief arguments advanced for their use. Strauss' opinion is that a more liberal though definite diet and a regulated manner of living have more permanent effects than the so-called short "cures." Since the course of the disease is essentially chronic, an endeavor should be made to regulate the diet and mode of life for an indefinite time. The selected diet should be both satisfying and nutritious and as like the usual diet of the patient as possible. In short, Strauss would prefer to treat the individual case of obesity rather than to treat by a definite plan. He gives some general rules. and tables of the caloric value of the different articles of food. He divides these into three classes—foods of high caloric value, those of poor caloric value, and those of moderate value. In a proper diet the protein content should be from 100 to 120 grams, which is equivalent to from 400 to 500 calories. In the Moritz milk cure the protein content is from 45 to 90 grams, which is equivalent to from 180 to 370 calories. The nitrogen-free food should be equivalent to from 1000 to 1500 calories. In the "quick cures" the nitrogen-free food is equivalent to less than 600 calories. The question whether the nitrogen-free food should be given in the form of carbohydrates or fats is a debatable one. Ebstein, Karell, Lenhartz, Tarnier, Schindler-Barnay, Debove-Gaval, Bouchard, and Moritz supply the bulk of the necessary calories by a preponderance of fats. In Hirschfeld's diet, the calories supplied by fats and carbohydrates are nearly equal. Bunting, Hart, Kisch, von Noorden, and others favor the use of carbohydrates to supply the necessary calories. Strauss favors the last method, since patients, as a rule, tolerate the reduction of fats more easily than that of carbohydrates, especially such carbohydrates as bread and potatoes. Furthermore, carbohydrates are most easily combined with the necessary protein. Strauss believes that certain procedures may be of value to reduce weight in certain cases of obesity. Among these are various modifications of the milk cure. Thus, Lenhartz has modified the usual Karell cure to hasten the loss of fat in the ordinary treatment of obesity. He gives 800 c.c. of milk for periods of from six to eight days. Roemheld uses 1000 c.c. of milk on two days of each week, combined with a restricted diet for the remainder of the week. Boas makes use of fast days, when patients receive tea with saccharin and lemon, fat-free bouillon, the whites of two or three eggs, a few sour apples, and approximately 100 grams of whole wheat or Graham bread. This diet contains about 470 calories. The Oertel treatment and also the various milk cures have the advantage of a restriction in the amount of fluids. Furthermore, the milk cures have a low sodium chloride content. Both of these tend to a loss of weight which is not due entirely to the loss of fat.

The tables which Strauss gives are valuable and offer a great variety of foodstuffs to choose from. Some general rules which he follows in diet are valuable. He uses meat in amounts not of more than 300 grams a day. The whites of hard-boiled eggs and fat-poor cheese may be added to supply the necessary amount of protein. Green vegetables

and various salads add variety to the diet. Potatoes are allowed, and are valuable because of their satisfying effect upon the appetite. Among the vegetables which he advises are green beans, spinach, cauliflower, asparagus, mushrooms, and the cabbage group. He forbids grapes, bananas, figs, dates, and nuts. Other fruits may be eaten raw or cooked with the addition of saccharin. Bread is given preferably in the form of whole wheat bread or diabetic bread containing albumen. Alcoholic beverages are forbidden. Other beverages should not be taken with meals. They should be restricted in amount and taken between meals. The intervals between feedings should not be long, and a cup of tea, bouillon, or some raw fruit taken between meals may lessen the appetite for the next meal. The sensation of an empty stomach is to be avoided, and this can easily be done by the use of some article of food poor in caloric value.

Tuberculosis.—WRIGHT (*Jour. Amer. Med. Assoc.*, 1909, lii, 1227) believes that mercury has a specific action in tuberculosis and gives some statistics to support this belief. He compares the results obtained by the routine treatment with the results obtained by the addition of deep muscular injections of mercury to the routine treatment. In a series of 161 cases, 83 patients voluntarily chose the addition of mercury to the routine treatment, while 78 patients preferred the routine treatment alone. Of the 161 cases, 8 were cured. Of those cured, 87.5 per cent. took the mercury treatment, while 12.5 per cent. took the routine treatment. There were 43 cases that showed marked improvement; all of these took the mercury treatment. Sixteen cases were improved; of these 87.5 per cent. had the mercury treatment, while 12.5 per cent. had the ordinary routine measures. Seventeen of the cases were classified as slightly improved; of these, 58.82 per cent. were on the mercury treatment, and 41.18 per cent. were on the routine treatment alone. Nineteen cases remained stationary; all of these took the routine treatment alone. There were 42 cases that showed an advance in the tuberculous process. Of these, 9.52 per cent. took the mercury treatment and 90.48 per cent. took the routine treatment. Sixteen deaths occurred in the series; of these, 31.2 per cent. were on the mercury treatment, and 68.8 per cent. were on the routine treatment. Combining the items cured, marked improvement, improved, and slightly improved, under the heading improved, stationary as such, and combining the items failed and died under the heading, failed, the following percentages are obtained. Of the 83 patients on mercury, 89.16 per cent. improved; none were stationary; 10.84 per cent. failed. Of the 78 patients on the routine treatment, 12.82 per cent. improved; 24.36 per cent. remained stationary; 62.82 per cent. failed. Wright gives the mercury by deep intramuscular injections in gradually increasing doses until the therapeutic limit is reached. After the maximum dose is established, it is then divided by two and the injections continued with this dosage. During the intervals between the injections the use of potassium iodide, which Wright formerly advised, is discontinued.

PEDIATRICS.

UNDER THE CHARGE OF
LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,
OF PHILADELPHIA.

Acquired Venereal Infections in Children.—F. POLLACK (*Johns Hopkins Hospital Bulletin*, 1909, xx, 142) states that yearly in Baltimore there are about 1000 cases of acquired venereal infection in children, thus proving that they are far more common than the medical profession or the laity realizes. The cause of most of the outrages upon children is found in the superstition that a person infected with either syphilis or gonorrhœa may get rid of it by infecting another, and preferably an "untouched virgin." Thus, a defenceless child is the most natural victim. Pollack has seen 189 such cases personally in the last six years. The infections run a milder course as regards complications than in adults; the duration of the disease is as long. The complications are as follows: Urethritis, 36; bubo, 28; secondary syphilis, 29; peritonitis, 19; bleeding, 16; primary sores, 7; pregnancy, 6; Bartholinitis, 3; arthritis, 3; ophthalmia, 3; etc. As treatment does not seem to influence the disease, prophylaxis seems to be the most effective remedy. In the secretions gonococci can be demonstrated very readily.

The Treatment of Facial Paralysis Due to Mastoid Disease or to the Mastoid Operation.—F. SYDENHAM (*Brit. Med. Jour.*, 1909, i, 1113) reports the case of a boy, aged five years, in whom, during an operation for mastoid disease, the facial nerve was divided, resulting in complete paralysis of that side of the face. Two days later the wound was re-opened and an end-to-end anastomosis performed; when apposed a gap of about one-half inch was noticed; this was bridged with silkworm gut. Electricity was employed from the first, but no movement of any kind could be produced for three months, when slight movements of the alæ nasi of the affected side could be observed upon forced sniffing; the other facial muscles afterward gradually returned to full power. The boy has now recovered his normal expression; when at rest and when his facial muscles are thrown into action, it is difficult to tell that there has been any facial paralysis at all. Sydenham recommends this method of anastomosis in preference to the faciohypoglossal and to the faciospinal accessory, as being more easily performed and better for the patient.

A Clinical Study of the Children of Tuberculous Parents.—J. A. MILLER and I. O. WOODRUFF (*Jour. Amer. Med. Assoc.*, 1909, lii, 1016) have made a study of 150 children of parents under treatment for pulmonary tuberculosis. The factors taken under consideration were the tuberculin test, pulmonary symptoms and signs, sputum examination, joint, bone, and other non-pulmonary lesions, the state of nutrition, hypertrophied cervical lymph nodes, tonsils, and adenoids. The following

are their conclusions: (1) In the children of tuberculous parents, who live in close association with such parents, a large proportion (in their series, 51 per cent.) become infected with tuberculosis. (2) The earliest manifestations are found in the lungs, and not in the superficial glands, bones, and joints. (3) The physical signs in children under ten years of age consist usually of persistent fine rales just without the midclavicular line in the fifth and sixth intercostal spaces; persistent sibilant rales in various parts of the chest make the diagnosis doubtful, but such cases should be regarded with suspicion. (4) The tuberculin tests, more particularly the hypodermic test in doses up to 5 mg., furnish the most reliable means of diagnosis. Of the local tests, the cutaneous is more reliable in children than the ophthalmic; it presents none of its possible dangers. (5) The ordinary examination of the sputum is almost no aid in the diagnosis of early tuberculosis in children. (6) Malnutrition is sometimes the only appreciable evidence of tuberculosis in children. (7) Hypertrophied tonsils and adenoids do not incline the balance in a suspected case toward a positive diagnosis of tuberculosis; further investigations of this subject, however, should be made. (8) The evidence that enlargement of the cervical lymph nodes is of aid as a determining factor in arriving at a diagnosis of tuberculosis in children is not conclusive.

Constitutional Eczema of Infants.—E. FEER (*Münch. med. Woch.*, 1909, lvi, 113) calls attention to chronic eczema occurring oftenest during the first few years of life and most commonly during the nursing period. In contradistinction to chronic eczema the acute form is frequently a true dermatitis; intertrigo is a true dermatitis only if coexisting with a disposition to eczema. The eczemas of older children rest oftenest upon a tuberculous base, and a positive tuberculin reaction may be obtained in almost every case. The real chronic eczema is undoubtedly a constitutional disease, but secondary dermal disturbance is quite common in their course. The two factors of importance in the development of chronic eczema are an inherited disposition and the type of nutrition; either of them may be the more important. There are two forms of chronic eczema to be found: the weeping, scale-forming eczema of the head, and the disseminated dry form. The first form is found oftenest in fat, pasty children, but even the healthiest may be affected. It begins in the scalp, spreading to cheeks and ears; it may spread to other parts of the body; there is very little itching. Overfeeding and constipation are common with these children and a spontaneous cure may be noted, when at the end of the first year a mixed diet is ordered for the child. The second form occurs almost exclusively in artificially fed infants; overfeeding is common and the patients are usually pale, thin, and flabby; chronic nutritional or intestinal disturbances are frequent. It manifests itself as discrete, dry, red, desquamating, infiltrating islands; also as papular and pustular foci. Weeping and scale formation are uncommon; part or all of the body may be involved. It is slow in development, itches, and is difficult to eradicate. Both the gouty diathesis and auto-intoxications have been blamed for this condition. Sudden deaths in children with such eczemas must be referred to "status lymphaticus" rather than to sepsis. Feer's

treatment consists of softening the scabs with oil or vaseline; this must be done gradually. He treats weeping surfaces with dilute solutions of aluminum acetate for several days, following it by a paste of oxide of zinc, starch, and vaselin. He bandages the hands to prevent scratching. Naphthalan is added in slowly healing cases, sulphur in the seborrhoic forms. Bismuth, zinc, or Lassar's salve is used in dry cases. Fowler's solution internally and slight laxation are of advantage. The amount of food must be reduced to a minimum, the quality also must be reduced. Barley water may be substituted for some of the breast feeding. A reasonable loss in weight need not be feared. Starchy food should be given in reasonable quantities to infants more than four months old; after the sixth month vegetables are allowed. Eggs and soups should be avoided under all conditions. The diet which has brought about the cure should be continued for months. In underfed children cutting down of diet is not permitted; the children should be fattened.

The Distribution of Bacteria in Bottled Milk.—Hess (*Deut. med. Woch.*, May, 1909) says that in bottled milk for children and babies, it should be considered that most of the bacteria are found in the upper layers of the cream, and become less in number in successive lower layers. The top 60 c.c. of cream holds the greater portion of the bacteria, and this holds good for the tubercle bacillus as well as streptococci and other germs. He believes that the top 60 c.c. of cream should be removed from milk intended for babies and children, instead of using the whole top part. He points out that a milk with 4.2 per cent. fat will still have 3 per cent. fat after taking the 60 c.c. from the top, and that this procedure is of benefit to the child.

The Early Symptoms of Anterior Poliomyelitis.—L. E. La Fetra (*Archives of Pediatrics*, May, 1909) has studied 63 cases of anterior poliomyelitis, to determine the more important early symptoms. Vomiting occurred in 25 cases, as a rule only at time of onset. Restlessness and irritability were common, and definitely noted in 37 cases. Tendon reflexes were absent in the paralyzed limbs in 16 out of 20 cases, and present, but sluggish, in 3. In no case in the whole series were tendon reflexes exaggerated. Cough, tonsillitis, or sore throat was noted in only 6 cases. Delirium was present in but 2 cases; convulsions in 4 cases. Rigidity of the neck occurred in 11 cases. Pain and tenderness in the affected limbs was present in 32 cases. Pain was in the muscles in 5 cases, on movement of joints in 4 cases. The latter is important, as possibly confusing the diagnosis with cerebrospinal meningitis and neuritis. The paralyzed limb was flaccid in 58 cases. It was spastic or rigid in only 5 cases. Paralysis came on early. It occurred on the first day in 24 cases; on the second day in 9 cases; on the third day in 3 cases; after two weeks in 4 cases. Paralysis was occasionally noted in the muscles of the neck, face, back, and abdomen. Also paralysis of the bladder and general anesthesia. Leukocytosis varied from 13,000 to 20,000.

OBSTETRICS.

UNDER THE CHARGE OF
EDWARD P. DAVIS, A.M., M.D.,
PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Ligation or Excision of Thrombosed Veins in the Treatment of Puerperal Pyemia.—WILLIAMS (*Amer. Jour. Obst.*, May, 1909) reports 5 cases of puerperal pyemia in which the veins of the broad ligaments were ligated or resected. In some of these cases an enlarged septic tube was removed; 4 of the 5 cases recovered, one of them being complicated by pneumonia and pleurisy. Williams has collected from the literature 15 cases of extraperitoneal operation, with 12 deaths—a mortality of 80 per cent.; 10 cases of transverse peritoneal operation, with a mortality of 70 per cent.; 5 cases of ligation or excision of both spermatic veins after laparotomy, with 60 per cent. mortality; 4 cases of ligation or excision of one spermatic or hypogastric vein, with 25 per cent. mortality; 2 cases of ligation of both spermatics and one hypogastric vein, with 50 per cent. mortality; 8 cases of ligation or excision of both spermatic and hypogastric veins, with 25 per cent. mortality; 2 cases of ligation of one hypogastric vein, with 100 per cent. mortality; 3 cases, in which no details are given, of ligation of the broad ligament veins, with 66.6 per cent. mortality. Williams concludes that the average mortality of puerperal pyemia is in the neighborhood of 66.6 per cent., which justifies any operation offering a chance of improvement. His paper is based upon the study of 56 cases of thrombophlebitis treated by the excision or ligation of one or more pelvic veins: 15 operations by the extraperitoneal method gave a mortality of 80 per cent.; 41 cases by the transperitoneal method gave a mortality of 43.9 per cent. This is no especial improvement upon expectant treatment. If, from these cases, those are deducted which were not susceptible of cure, and in which the technique was faulty, the corrected mortality for extraperitoneal operation was 40 per cent., and for transperitoneal operation 21.4 per cent. In Williams' 5 cases the gross mortality was 20 per cent. Early operation for thrombosis limited to the spermatic vein should not give a mortality exceeding 10 per cent., as compared with 25 per cent. when other vessels are involved. A positive diagnosis can be made when a worm-like mass can be palpated at the outer portion of the broad ligament in patients suffering from chills and hectic temperature. Operation should then be undertaken. Excision of the thrombosed vessels is rarely necessary, and when the vessel appears likely to rupture or is surrounded by periphlebitic inflammation, excision should be undertaken; otherwise ligation only should be performed. The transperitoneal is preferable to the extraperitoneal method. The transperitoneal operation is easier, gives a better view of the vessels, and scarcely increases the danger of peritoneal infection. Laparotomy should be done in all cases in which interference seems indicated, because the vaginal route is applicable only to a small class of cases in which thrombosis is limited to the vessels of the broad ligament

exclusively. A diagnosis of this condition can rarely be positively made.

Exophthalmic Goitre Complicating Pregnancy.—STOWE (*Amer. Jour. Obst.*, May, 1909) reports a fatal case of exophthalmic goitre complicating pregnancy. The patient was aged twenty-four years, and had the appendix removed at the age of twenty-one. At this time the peritoneum was the seat of miliary tuberculosis. She afterward aborted at two and a half months, probably due to the goitre, as the symptoms had made their appearance a few months before. When pregnancy again occurred the characteristic signs of goitre were present. The thyroid gland was slightly enlarged, chiefly on the right side. Under rest in bed and appropriate treatment, the patient's general condition improved, but the ocular signs and enlarged gland remained unchanged. Shortly after pregnancy occurred the patient vomited incessantly for eleven days, and her condition rapidly became desperate from acute toxemia. The uterus was emptied so far as possible, under ether, but the patient died in coma three days afterward. An autopsy could not be obtained.

Symphysiotomy.—PRENTISS (*Amer. Jour. Obst.*, May, 1909) reports the case of a white woman whose first labor terminated after a very difficult forceps extraction, with the death of the child. The pelvis was symmetrically contracted 2 cm. in each diameter. At the eighth month of pregnancy rapid dilatation was performed under ether, the forceps applied above the superior strait, and traction made for about one-half an hour when the head failed to descend. The pubic joint was then opened, the bones separating 6 or 8 cm., the child being readily delivered. No injury to the bladder, urethra, or vagina occurred, and the bones were wired together. The patient died suddenly on the third day, after being moved. At autopsy no evidence of sepsis was present, but thrombosis had developed in the veins of the uterus, left broad ligament, left ovarian vein, and right heart. The child survived. This case draws renewed attention to the danger of unsuccessful attempts at forceps delivery.

The Clinical Value of Opsonins in Pregnancy and the Puerperal State.—GUGGISBERG (*Zeitschr. f. Geb. u. Gynäk.*, 1909, lxiv) reports the results of his experiments in pregnant and parturient women. The spontaneous phagocytosis of the human leukocytes is present in staphylococcic and streptococcic infection. In normal human serum substances are found which increase the phagocytosis of the staphylococci and strepcococci through the cells of the human tissue. In pregnant patients the opsonic index is not the same, and varies in a number of pregnant patients, as regards both bacteria, from the average of the normal human being. In some pregnant patients the opsonic index is normal. In the puerperal period the opsonic indices for staphylococci and streptococci are not similar, but vary greatly in different patients. It was impossible to estimate precisely the cause of this variation. As a general result of his investigations Guggisberg could not find a definite value for the opsonic index in prognosis and diagnosis in septic infections of pregnant and parturient women.

The Results of Hebostiotomy from the Study of Seven Hundred Cases.—Schläfli (*Zeitschr. f. Geb. u. Gynäk.*, 1909, lxiv) obtained the following results in the study of 700 hebostiotomies: In 510 cases there was considerable hemorrhage during the operation in 10.98 per cent.; the bleeding became profuse in 2.94 per cent., and abundant in 1.37 per cent. Taken in all, hemorrhage was greater than normal in 15.3 per cent. In 664 cases, hemorrhage occurred during operation in 0.3 per cent.; in 510 cases there was hematoma or laceration communicating with the vagina in 15.49 per cent. This was most frequently observed among primiparæ in 32.5 per cent.; among multiparæ less frequently, 11.88 per cent. Simple lacerations of the birth canal occurred in 17.84 per cent. The mortality of those patients having lacerations communicating with the vagina was 12.66 per cent. A fatal result could be directly traced to the lacerations in 31.25 per cent. In fatal cases, lacerations of the genital tract were a potent factor in 40.6 per cent. Injuries to the bladder occurred in 12.35 per cent.; fever during the puerperal period in 31.76 per cent.; and thrombophlebitis in 8.23 per cent. There was hernia between the edges of the cut bone in 7.5 per cent. after the operation; prolapse of the vagina in 24.17 per cent.; and incontinence of urine in 4.17 per cent. The corrected mortality among the mothers following the operation was 4.37 per cent.; the corrected mortality for the children was 9.18 per cent.

Correlation of the Uterus and Ovaries.—Bell and Hicks (*Brit. Med. Jour.*, March 13, 1909) report, from their studies of the correlation of the uterus and ovaries, that the removal of the ovaries during pregnancy was followed invariably by abortion. The internal secretion of the ovary is evidently favorable to the continuation of pregnancy—because of the relation between the ovary and the calcium metabolism of the body. In the human subject, however, this secretion is not absolutely essential in all cases.

Elective Cesarean Section.—Porter (*Jour. Amer. Med. Assoc.*, March 20, 1909) has collected the report of 126 cases of elective Cesarean section performed by thirteen American operators upon uninjured, uninfected, and unexhausted women. The maternal mortality of the series was 1.58 per cent., with no fœtal mortality. The maternal mortality of the high forceps and version is stated at 1.14 per cent., with a fœtal mortality of 17.3 per cent. The maternal morbidity of Cesarean section in this series was 12.69 per cent., and that of high forceps or version, 42 per cent. The fœtal morbidity in Cesarean section was nothing, compared with the fœtal morbidity of high forceps and version of 12.2 per cent. Porter urges that in contracted pelves Cesarean section is the operation of choice, especially in primiparæ. If the child is viable Cesarean section should be performed in central placenta prævia. In eclampsia, at or near term, in a primipara, abdominal Cesarean section should be chosen, and when the child is small, and the vagina and pelvis capacious, vaginal Cesarean section should be selected. In cases requiring abdominal section at term, or the removal of uterine ovarian tumors, the child should be delivered by Cesarean section immediately before or after the removal of the tumor. In elderly primiparæ at term, with a vigorous child, a normal pelvis, but

rigid soft parts, unusually sensitive to pain, in bad physical condition, and with bad nervous equilibrium, early Cesarean section offers the best chance for life and health to mother and child. The fact that Cesarean section entails no risk upon the child should cause the operation to be limited to those cases in which the child is in good condition.

GYNECOLOGY.

UNDER THE CHARGE OF

J. WESLEY BOVÉE, M.D.,

PROFESSOR OF GYNECOLOGY IN THE GEORGE WASHINGTON UNIVERSITY, WASHINGTON, D. C

Menstruation and Menorrhagia.—FLORENCE E. WILLEY (*Jour. Obst. and Gyn. Brit. Emp.*, 1909, xv, 236) says we may still regard the monthly cycle as a preparation of the uterus for the development of an impregnated ovum, but we must realize that the important physiological requirement is an efficient blood supply to the uterine musculature. The changes in the mucosa are such as would occur in any congested state of the uterus with overfilled capillary vessels, and are probably secondary, the primary changes being in the muscular tissue. Anatomical studies of the capillary system of the uterus during menstruation and during the time of rest show that parts of this system are functional, that is, many capillary fissures are closed during the intermenstrual period and become filled with blood, and act as part of the general vascular system during menstruation and pregnancy. Clinical observation shows that the uterus increases in size during menstruation. Willey also says that uterine muscle possesses, in addition to its function of contractility, a possibility of hypertrophy unknown in any other muscle. Assuming all the above is true, Willey then suggests that menorrhagia should direct attention to the blood supply to the muscular wall of the uterus rather than to changes in the endometrium. Therefore, in the treatment of those cases of menorrhagia, in which newgrowths of the uterus or tubal disease may be excluded, general and local conditions that bring about uterine congestion or inefficient action of the uterine muscle must be sought. Massage of the uterus is condemned.

Tuberculous Peritonitis.—BROUN (*Amer. Jour. Obst.*, 1909, lix, 812) discusses the various factors in the etiology of tuberculous peritonitis especially mentioning the dictum of J. B. Murphy that the chief avenue of infection is through the Fallopian tubes and from coitus with tuberculous men. From Leuret the following conclusions are adopted: (1) Genital lesions are the most frequent cause of peritoneal tuberculosis in women, in particular the clinical type known as idiopathic ascites in young women. (2) Tuberculosis of the adnexa is always accompanied by tuberculous peritonitis; the form occurring being one of two types—ascitic peritonitis, free or encysted, and dry pelvic peritonitis with adhesions. (3) The type of pelvic peritonitis with adhesions is

subject to frequent exacerbations. In the surgical treatment of this lesion Broun recommends ablation of both Fallopian tubes, and, if suspected, after careful palpation and inspection, the vermiform appendix. Complete closure of the abdominal wound without drainage is advocated. With hygienic after-treatment the use of tuberculin is advised.

Aneurysm of the Uterine Artery.—Reymond (*An. de gyn. et d'obst.*, 1909, xxxvi, 117) presented to the Société Anatomique de Paris a specimen of aneurysm of the uterine artery removed by an abdominal operation. The symptoms were those of cancer of the corpus uteri after the menopause. To touch, the uterus was found displaced slightly to the left and the pulsations of the aneurysm in the right lateral part of the posterior fornix vaginæ were evident. The aneurysm extended from the origin of the uterine artery to the uterine cornu, and was exsected. The dilatation was the size of the thumb. Hemorrhage did not occur after the operation.

Resection of Seventy Inches of Intestine after Perforation of the Uterus.—Barton and Smalley (*Jour. Amer. Med. Assoc.*, 1909, lii, 1665) add another to several cases recently appearing in medical literature of perforation of the puerperal uterus and pulling through the opening a considerable length of the intestine. The case reported was one of perforation of the uterus with ovum forceps in using them to complete an abortion. Barton and Smalley were at once called and opened the abdomen, encountering much free fluid, blood, and a hole one and a half inches wide through the uterus. From the uterus the incarcerated intestine was drawn back into the peritoneal cavity. A four or five months fœtus and placenta were then withdrawn from the uterine cavity by enlarging the opening. A portion of the jejunum and of the ileum, seventy inches in length was found to be detached from its shrunken and black mesentery. It was resected and an end-to-end anastomosis of the intestine was made successfully. An interesting incident was that a long-standing intestinal indigestion has not recurred during the eighteen months elapsed since the operation.

Treatment of Myoma.—Kurt Torkel (*Monatsschr. f. Geburtsh. u. Gynäk.*, 1909, xxxi, 338) reports 303 cases of myoma observed at Breslau during 1902-1908. A study of these cases shows that radical measures are not invariably indicated, while, on the other hand, too much reliance should not be placed on palliative measures which may unnecessarily prolong the condition. Curettage is indicated in suitable cases, but atmocausis and internal medication are to be denounced. Operative intervention should always meet with the requirements of the individual case. With the exception of special cases, conservative methods are of value only when retention of the uterus would prove of value to the patient. In all other cases, as a rule, supravaginal amputation should receive preference over abdominal total extirpation.

The Diagnosis of Tuberculous Peritonitis.—J. N. West (*Amer. Jour. Obst.*, 1909, lix, 820) states that in fully one third of the cases of tuberculosis of the peritoneum operated upon the previous diagnosis was

ovarian cyst, and that in another third the diagnosis was pyosalpinx of the other forms. Quoting C. H. Mayo, West states that in 78 per cent. in the United States and in 89 per cent. in Germany of all cases in which necropsies have been held, there is shown the presence of, or the evidence of, past existence of tuberculosis. Of 13,922 necropsies collected by Grawitz and Brun, 2 per cent. showed tuberculous peritonitis. The proportion of women to men as victims of tuberculous peritonitis is said to be 5 to 1, and the most common period of infection is from twenty to thirty years. The chief points given to establish a clinical diagnosis are recurrent attacks of pelvic peritonitis extending over several months; gradual accumulation of fluid in the abdomen; hectic, afternoon or evening, temperature; gradual and progressive loss of weight and strength, and sensitiveness of the abdomen. The clinical diagnosis failing, the aid of other tests may be applied. Of these the skin inoculation test of von Pirquet, is preferred to that of the Calmette eye installation or the skin inunction test of Moro. While these are not infalliable tests, the data thus obtained are valuable. Curiously enough, when considered superficially, the absence of reaction is more reliable than the occurrence of a reaction.

Perforation of the Stomach and Small Intestine as a Sequel to Ovariotomy and Hysterectomy.—J. BLAND-SUTTON (*Jour. Obst. and Gyn. Brit. Emp.*, 1909, xv, 197) reports three cases of perforation of the stomach or the small intestine after ovariotomy or hysterectomy that have come under his observation. Two of them died on the eighth day after operation. The third died forty-six hours after hysterectomy. Bland-Sutton refers to casual reference to the personal experience of von Rosthorn and Olshausen with this complication, and appeals to gyncologists to report similar experiences.

Hernias through the Pelvic Floor.—BARRETT (*Amer. Jour. Obst.*, 1909, lix, 553) believes that in the treatment of hernias of the pelvic floor consideration should be given to the following principles: (1) The abdominal organs need for support, in addition to their own ligaments, a limiting wall. (2) The limiting wall should grow stronger from above downward, as gravity of the organs exerts a force not exerted above. (3) The pelvic floor, being the lowest part of the abdominal wall, needs to be strongest. (4) Abdominal organs herniate through the walls by reason of increased pressure, insufficient supports, and weak places in the walls or "faults." (5) The pelvic floor, does not act as a direct support to the uterus, as the uterus does not lie against it. (6) The pelvic floor by reason of its importance in man has developed an additional diaphragm—the levator ani muscle and its fascia. (7) This diaphragm is weakened by "faults" (the rectum, vagina, and urethra). (8) The vaginal canal, by reason of extreme dilatation which lacerates the surrounding levator muscle, becomes a frequent site for hernia. (9) Atavism of the levator ani muscle occasionally furnishes a congenital predisposing cause of hernia through the rectum in the male, and the rectum and vagina in the female. (10) The cure of these hernias, due either to traumatism or congenital defects, should look toward, (*a*) lessening intra-abdominal pressure; (*b*) correcting the pelvic floor defect; (*c*) putting the vagina out of a vertical line;

(*d*) putting the uterus out of line with the vagina. (11) The posterior segment of the pelvic floor is repaired by uniting the levator ani muscle and its fascia, and the lower end of the vagina is thereby pushed forward. (12) The anterior segment may be corrected by anterior colporrhaphy and, if occasion demands, a shortening of the sacro-uterine ligaments. The upper end of the vaina is thus pushed backward. (13) The uterus is got out of line with the vagina, (*a*) by shortening the round ligaments; (*b*) by vaginofixation in cases of mild prolapse past the menopause; (*c*) by vaginal hysterectomy in cases of marked prolapse, especially with a pathological uterus; in case of hysterectomy the ligament should be implanted into the vaginal vault; (*d*) by bringing the lower end of the broad ligaments in front of the cervix, according to Alexandroff, which operation is slightly modified in this country by Dudley and others. Barrett emphasizes that the pelvic floor is an important factor in abdominal support and that the chief structure is the levator ani muscle; that the tendency of the vagina to become a hernial canal is greatly increased by traumatism and congenital defects. In slight cases rest and lessened intra-abdominal pressure are recommended; in exaggerated cases repair of the pelvic floor, lessening of the vertical position of the vagina, removal of the uterus, or increasing its angle to the vagina should be done.

OTOLOGY.

UNDER THE CHARGE OF

CLARENCE J. BLAKE, M.D.,

PROFESSOR OF OTOLOGY IN THE HARVARD MEDICAL SCHOOL, BOSTON.

Ear Disorders in Diseases of the Urogenital Apparatus.—Dr. J. Sendziak (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 55), presents an interesting summary of the several ear affections that may complicate diseases of the genito-urinary organs. Considering first the kidneys, he points out the occasional occurrence of hemorrhages into the tympanum or labyrinth in the course of severe acute or chronic parenchymatous nephritis; sometimes these hemorrhagic spots can be observed on the drumhead, particularly in the anterior upper portion. The clinical symptoms, which suggest an aural examination in nephritis, are sudden defects of hearing, subjective sounds, and giddiness. He lays especial stress upon the frequency with which purulent otitis media is associated with that form of nephritis that follows scarlet fever, pointing out a prognostic significance of this connection; it having been repeatedly observed that an increase in the otorrhœa accompanies an exacerbation of the kidney trouble, and vice versa. Regarding the relationship between aural affections and diseases and abnormalities of the genital organs, he finds such instances much more frequent among females than males. Vicarious menstruation, when hemorrhage from the ear sub-

stitutes the normal discharge, is said to have been noted six times in 200 vicarious cases. The appearance of a mild dermatitis of the auricle or canal at the monthly period, or during pregnancy has been often observed, and the stubbornness of such inflammations when occurring about the climacterium is dwelt upon. Perhaps the most important point mentioned in the paper refers to the unfavorable influence sometimes exerted upon aural affections by pregnancy and childbirth; any existing pathological process in the ear may suffer a considerable exacerbation during the period of gestation, and only exceptionally does there follow a reclamation of the hearing power lost in such cases.

A Pure Transudate in the Middle Ear.—WALB, of Bonn (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 317), refers to the discussion which took place at the last session of the German Otological Society, at Bremen, as to the possibility of a sterile exudate or transudate into the tympanic cavity occurring. He holds, with Kümmel, Schiebe, and others, that there are many instances of pure intratympanic exudate, resulting from negative intratympanic pressure, and that such fluids, naturally, are sterile. In support of his contention, he cites clinical cases in which the middle ear disturbance arises from closure of the tympanopharyngeal tube, and in which an incision of the tympanic membrane disclosed the following conditions: Sterile fluid evacuated; the tympanic mucous membrane presenting characteristic appearances of tissue under negative pressure; an incision that remained sterile, even though unhealed, for weeks; and, in some cases wherein the causes of negative pressure had not been removed, a recurrence of the transudate shortly after healing of the incision. Walb's views coincide with the commonly accepted views here that the simple exudative otitis medias, occurring without inflammatory symptoms, usually in elderly persons or in those who have stricture or obstruction of the tympanopharyngeal tubes, are not of bacterial, but rather of mechanical, origin.

The Surgery of the Labyrinth of the Ear.—During the past five years operations upon the labyrinth have been quite frequently performed, and the revived study of the physiology and anatomy of this portion of the organ of hearing, incited by these surgical investigations, together with the clinical experience resulting therefrom, is giving a broader knowledge of the internal ear affections and holding out some promise of a more enlightened treatment in the future of these previously ignored or neglected conditions. It was quite natural to pursue further the excellent surgical work that has developed in the treatment of suppurative otitis media, and to open the semicircular canals or the cochlea when, in the course of a mastoidotympanal exenteration, they were found to be diseased. The symptoms of labyrinthine invasion associated with purulent otitis media are not yet so clearly defined as to make it always possible to diagnosticate the condition prior to operation, but great progress is being made in that direction. Meanwhile, all cases of chronic suppurative otitis that present evidences of labyrinthine disturbance, such as frequent or continued headaches, severe tinnitus, nausea and vomiting, or great deafness, are being carefully investigated during the course of the operative intervention that is necessary to cure the middle-ear disease.

In an interesting paper on "Indications for Labyrinth Opening in Complicated Middle-ear Suppurations and New Suggestions as to Methods," W. UFFERNORDE (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 227) discusses these questions at some length. He points out the fallibility of all known tests to indicate invasion of the labyrinth by purulent processes, and the dangers of operating in this region; so great is the risk, and so frequently do what were apparently symptoms of labyrinthine disease disappear after eradication of the disease from the middle ear and mastoid cells, that he cautions against needless or unnecessary operations upon the internal portion of the ear. Even when carious openings in the bony wall of the semicircular canals are discovered during an operative exploration, it must be remembered that this process may be superficial, may not yet have broken through the membranous capsule, and so a curettement of the labyrinth may not be called for. In the cases of pronounced purulent labyrinthitis the results of surgical treatment have been brilliant; such conditions are practically always fatal unless treated by operation. The difficulty at present lies in determining which cases shall be submitted to operation.

Much has been recently reported concerning the clinical value of the caloric tests of equilibrium, and it might be supposed that these, added to von Stein's tests (walking, jumping, rotation, etc.), would give certain rules by which to ascertain the location and extent of a labyrinthine lesion. Uffernorde discourages dependence upon these tests, while encouraging further study of them, and says that he has seen all the rules clinically reversed. If at the present the situation seems to be somewhat doubtful, it is certainly hopeful and there is prospect of important discoveries in this field. It will not be surprising if in the very near future the labyrinth of the ear becomes quite as much an open book to the otologist as is the tympanic cavity now.

In this connection it is interesting to note that much attention is being given to the study of aural vertigo and tinnitus aurium. These are conditions that have long baffled observers and about which very little is known with certainly. MATTE (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 142), in an article on "The Surgery of the Labyrinth," considers an operation for the cure of distressing tinnitus. He recites the character of the serious type of this affection, the noises sometimes being so serious and persistent as to drive the victim to the contemplation of suicide, and recommends in such cases operative ablation of a portion of the labyrinth. He believes that by a careful study of the nature of the sound heard, and comparison of it with other measurable sounds under control of the investigator, it may be possible to localize the lesion, and thus determine what part of the vestibule or cochlea to attack. He reports a successful operation of partial ablation of the labyrinth, the details of which are interesting as showing what may be conservatively effected. Section of the auditory nerve has been successfully done for the relief of subjective sounds, but this totally destroys hearing, while Matte's suggestion, if it further proves to be practicable, may serve to relieve the patient of a distressing affection without completely destroying the hearing.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
WARFIELD T. LONGCOPE, M.D.,
DIRECTOR OF THE AYER CLINICAL LABORATORY, PENNSYLVANIA HOSPITAL,
ASSISTED BY
G. CANBY ROBINSON, M.D.,
CLINICAL PATHOLOGIST TO THE PRESBYTERIAN HOSPITAL, PHILADELPHIA.

The Action of Narcotics and Alkaloids on the Complement.—GOLDSCHMIDT and PRIBRAM (*Ztschr. f. exp. Path. u. Ther.*, 1909, vi, 211) have studied the action of various narcotics and alkaloids on hemolytic sera and have obtained the following results: The narcotics of the aliphatic and aromatic groups as well as powerful brain and nerve poisons in general (especially the alkaloids) exert a group of actions which stand in causal relationship to one another. (1) The action on lecithin suspensions as well as on other colloidal suspensions is either solution or precipitation; subsequent solution of the precipitate may take place. (2) Fresh hemolytic sera lose their power to destroy red blood cells when subjected to the action of the above-mentioned poisons. This action is due to a change in the physical condition of the medium and can be reversed by again adding sufficient fresh serum. The phenomenon is probably similar to the inactivation by heating to 60° C., since both procedures are accompanied by lowering of the surface tension. (3) Red blood cells are laked by powerful brain and nerve poisons with few exceptions. (4) The action of erythrocytic and cerebral bacterial toxins (tetanus, diphtheria) is weakened or prevented by these poisons. (5) All of these actions are of physical nature and are due to the intense physical activity of the above-mentioned substances. (6) Only poisonous alkaloids produce this phenomenon, and they probably stand in close relationship to their chemical constitution.

The Origin of Nephritic Œdema.—JULIUS BENCE (*Zeit. f. klin. Med.*, 1909, lxvii, 69), in order to elucidate certain problems in connection with œdema in nephritis, studied the fluid content of the blood in normal and nephrectomized rabbits, and in rabbits in which a nephritis had been produced by injections of uranium nitrate. When rabbits whose kidneys are normal are deprived of water, there is a slow but constant loss in weight with a decrease in the fluid content of the blood. If such rabbits are given sufficient water by the stomach to keep the weight at a constant level, that is, from 100 to 150 grams of water a day, the fluid content of the blood is not perceptibly altered. Nephrectomized rabbits when deprived of water also show a gradual loss in weight, but present interesting changes in the fluid content of the blood. At first this rises slightly, then falls temporarily, but finally increases steadily until the animal dies. With the hydremia, which continues in spite of the fall in body weight, œdema may develop, since three of the eight nephrectomized animals showed ascites. The ingestion of 100 to 150 grams of water a day, sufficient to keep the body weight at practically a constant

level, gives rise to a more pronounced hydremia and uniformly to œdema. Rabbits in which a nephritis has been produced by means of uranium nitrate, deprived of water, lose weight slowly during the first day; on the second day there is a sharp fall in weight synchronous with the diuresis produced by the nephritis. On the third day anuria makes its appearance and the body weight remains about the same. A drop in the fluid content of the blood accompanies the diuresis, but with the onset of anuria hydremia sets in and increases during the production of œdema and until death. In three of the eight animals there was ascites. Injection of water in the same amounts as was used in the other experiments gave rise to more hydremia, which started before the development of anuria, and was accompanied constantly by œdema. Uranium nitrate injected into nephrectomized animals did not influence the course of events, which seemed to show that the effects obtained by the use of chemicals are not due to injury to the vessel walls, but simply to the disturbance which it sets up in the kidney. The experiments show that when the kidneys are removed or made functionless through disease, there is an interchange of water from the tissues into the vessels giving rise to hydremia. A second interchange of fluid takes place between the blood and tissue spaces or serous cavities. These phenomena occur even though there is no addition of water to the body, though if water is obtained from outside the body, the effects, that is, the œdema, are much more marked.

The Relation of Typhoid Bacilli to the Gall-bladder.—Another e idence of the importance of the role played by bacillemia in typhoid fever is afforded by the experimental work of CHIAROLANZA (*Ztschr. f. Hygiene u. Infektionskr.*, 1908, lxii, 1) on the relation of typhoid bacilli to the gall-bladder and gall-ducts. He found that if bacilli are injected into rabbits subcutaneously, they do not reach the gall-bladder; but after intravenous injection they were found in 17 out of 23 cases (74 per cent.) The shortest time for them to reach the gall-bladder was two hours; the longest period of their excretion from it, fifty-eight days. To determine in what way they entered the gall-bladder, the cystic duct was ligated shortly before injecting the bacilli. In these cases also, bacilli were found in the bladder, showing that they had entered not with the bile, but through the capillaries of the mucosa and submucosa. Also, if the common duct was ligated, bacilli could be found in the intestine, showing that here also they might invade by way of the capillaries. On histological examination the infected gall-bladder showed degeneration of the epithelium in acute cases; proliferation of the epithelium with thickening and infiltration of the submucosa, in the more chronic forms. Necrosis of the liver was never found.

Notice to Contributors.—All communications intended for insertion in the Original Department of this JOURNAL are received only *with the distinct understanding that they are contributed exclusively to this* JOURNAL.

Contributions from abroad written in a foreign language, if on examination they are found desirable for this JOURNAL, will be translated at its expense.

A limited number of reprints in pamphlet form, if desired, will be furnished to authors, *provided the request for them be written on the manuscript.*

All communications should be addressed to—

DR. A. O. J. KELLY, 1911 Pine Street, Philadelphia, U. S. A.

CONTENTS.

ORIGINAL ARTICLES.

Intestinal Perforation during Typhoid Fever in Children 625
By JOHN H. JOPSON, M.D., Associate in Surgery in the University of Pennsylvania; Surgeon to the Presbyterian, the Children's, and the Bryn Mawr Hospitals, Philadelphia; and J. CLAXTON GITTINGS, M.D., Instructor in Pediatrics in the University of Pennsylvania; Visiting Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Medical Dispensaries of the Children's and the Presbyterian Hospitals, Philaphia.

The General Movement of Typhoid Fever and Tuberculosis in the Last Thirty Years 642
By GEORGE M. KOBER, M.D., LL.D., Professor of Hygiene in the Georgetown University, Washington, D. C.

Experiments Relating to the Bacterial Content of the Feces, with Some Researches on the Value of Certain Intestinal Antiseptics 653
By JULIUS FRIEDENWALD, M.D., Professor of Gastro-enterology in the College of Physicians and Surgeons, and T. FREDERICK LEITZ, M.D., Associate in Gastro-enterology in the College of Physicians and Surgeons, Baltimore, Md.

A Case of Carcinoma on Diverticulitis of the Sigmoid 661
By H. Z. GIFFIN, M.D., Physician to St. Mary's Hospital, and LOUIS B. WILSON, M.D., Director of the Laboratories of St. Mary's Hospital, Rochester, Minn.

Cervical Rib and its Relation to the Neuropathies 666
By S. P. GOODHART, PH.B., M.D., Chief of the Neurological Clinic, Out-patient Department, Mt. Sinai Hospital; Neurologist to the New York Red Cross Hospital; Junior Attending Physician to the New York Hospital for Nervous Diseases.

The Interpretation of Aphasia 683
By F. X. DERCUM, M.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College, Philadelphia; Neurologist to the Philadelphia General Hospital; Membre Correspondant Étranger de la Société de Neurologie de Paris.

School Life and its Relation to the Child's Development 702
By THOMAS MORGAN ROTCH, M.D., Professor of Pediatrics in the Harvard Medical School, Boston.

Compression of the Pulmonary Veins, the Pressure Factor in the Etiology of Cardiac Hydrothorax 712
By GEORGE FETTEROLF, A.B., M.D., Demonstrator of Anatomy in the University of Pennsylvania; Laryngologist to the White Haven (Pennsylvania) Sanatorium and the Henry Phipps Institute, Philadelphia; and H. R. M. LANDIS, A.B., M.D., Visiting Physician to the White Haven (Pennsylvania) Sanatorium and the Henry Phipps Institute; Demonstrator of Medicine in the Jefferson Medical College, Philadelphia.

A Practical Hospital Polygraph 727
By THEODORE B. BARRINGER, JR., M.D., of New York.

Locomotor Ataxia and Paralysis Agitans in the Same Patient . . . 729
By AUGUSTUS A. ESHNER, M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine.

A Case of Cerebral Tumor Presenting a Very Unusual Clinical Course 733
By R. D. RUDOLF, M.D. (EDIN.), M.R.C.P. (LOND.), Professor of Therapeutics in the University of Toronto, and J. J. MACKENZIE, B.A., M.B. (TOR.), Professor of Pathology in the University of Toronto.

REVIEWS.

A System of Medicine. By Many Writers. Edited by Sir Clifford Allbutt, K.C.B., and Humphry Davy Rolleston, M.D. 740
Surgery, its Principles and Practice. By Various Authors. Edited by William Williams Keen, M.D., LL.D., and John Chalmers Da Costa, M.D. 742
Diseases of the Digestive Canal. By Paul Cohnheim, M.D. 744
A Text-book on Embryology. By Frederick Randolph Bailey, A.M., M.D., and Adam Marion Miller, A.M. 746
Die Wurmfortsatz-Entzundung: eine pathologisch-histologische und pathogenetsche Studie. (Appendicitis: A Pathologico-histological and Pathogenetic Study.) By L. Aschoff 747
Self-control and How to Secure it. By Dr. Paul Dubois 749
A Text-book of the Diseases of the Ear for Students and Practitioners. By Professor Dr. Adam Politzer 749
New and Non-official Remedies 750

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D., AND W. S. THAYER, M.D.

Toxic Meteorism in Infectious Diseases 751
Abdominal Symptoms at the Onset of Pneumonia 752
The Leukocytes and Differential Count in Acute Abdominal Infection . 752
The Intravenous Injection of Diphtheria Antitoxin 752
The Opsonic Index in Typhoid-bacilli Carriers 753
The Measurement of the Blood Pressure 753
The Examination of the Eye-grounds in the Differential Diagnosis between Pernicious Anemia and Severe Secondary Anemia 754
Nodal Bradycardia 754
Clinical Symptoms of Hypertrophy of the Left Ventricle 754
The Rise of Blood Pressure in Nephritis 755
Effect of the Injection of Bile on the Circulation 755
The Influence of the Thyroid on Carbohydrate Metabolism 755

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D., AND T. TURNER THOMAS, M.D.

Transplantation of Joints 756
Experimental Investigations on the Sensibility of the Abdominal Cavity . 757
An Operative Cure of a Hernia into the Fossa Duodenojejunalis of Treitz . 758
Reduction of an Unreduced Dislocation of the Shoulder by Posterior Arthrotomy 758
Ligation of the Veins in Portal Thrombosis from Appendicitis . . . 759
The Operative Treatment of Tuberculosis of the Vas Deferens and of the Seminal Vesicles 759
The Late Results of Ureterocystostomies 760
The Double Filigree Operation for the Radical Cure of Inguinal Hernia . 761

THERAPEUTICS.

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.

The Karell Milk Cure: Its Modifications and Indications 762
Calcium Lactate in the Treatment of Epilepsy 763
The Use of Cerium Oxalate for the Relief of Vomiting 763
The Treatment of Fibrinous and Serofibrinous Pleuritis 764
The Employment of Iodopin in Syphilis 765
A Contribution to Digitalis Therapy 766
The Therapeutic Action of Potassium Bitartrate. 766

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.

The Relation of Infectious Diseases (Measles, Whooping Cough, and Influenza) to Tuberculosis in Children 767
The Incidence of Tubercle Bacilli in New York City Milk 767
Chronic Glomerulonephritis Treated by Double Decapsulation . . . 768
Napkin-region Eruptions in Infants 768
Rheumatoid Arthritis 769
Purpura Fulminans 769

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.

Repeated Cesarean Section and its Complications 770
The Treatment of Sepsis with Bacterial Vaccines 771
Accidental Perforations of the Uterus and Vagina 771
Symphysiotomy 772
Differential Diagnosis between the Hemolytic Streptococci 772

GYNECOLOGY.

UNDER THE CHARGE OF

J. WESLEY BOVÉE, M.D.

Complete Restoration of Genital Function in Gynatresias by Abdominal Operation 773
Amputation of the Uterus in the Corpus to Preserve the Menstrual Function 773
Contributions to the Clinic of Gynatresia 773
The Choice of Time for Operation for Pelvic Inflammation of Tubal Origin 774
The Involuntary Muscle Fibers of the Pelvic Floor 774
Indication for Operation in Chronic Inflammatory Adnexal Disease . . 775
Wertheim's Panhysterectomy for Carcinoma of the Cervix 775
Dry Heat as a Therapeutic Factor in Gynecology 775
Suture of Recent Perineal Tears 775

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

J. SOLIS-COHEN, M.D.

Regeneration of a Vocal Cord after Total Extirpation for Cancer . . . 776
Intranasal Tuberculin Reaction 776
Epithelioma of the Rhinopharynx 776
A New Pharyngoscope 777
Lipoma of the Larynx 777
Laryngostomy 777

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

WARFIELD T. LONGCOPE, M.D.,

ASSISTED BY

G. CANBY ROBINSON, M.D.

The Pathogenesis of Rabies 778
Changes in the Hypophysis Cerebri during Pregnancy 778
The Influence of Cardiac Insufficiency on the Disposition of the Blood in the Organs 779
Hypophysis Tumor and its Bearing on Acromegaly 780